

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:sss189dxw

PASSWORD:

LOGINID/PASSWORD REJECTED

The loginid and/or password sent to STN were invalid.
You either typed them incorrectly, or line noise may
have corrupted them.

Do you wish to retry the logon?

Enter choice (y/N):

Do you wish to use the same loginid and password?

Enter choice (y/N):sss189dxw

LOGINID:

PASSWORD:

LOGINID/PASSWORD REJECTED

The loginid and/or password sent to STN were invalid.
You either typed them incorrectly, or line noise may
have corrupted them.

Do you wish to retry the logon?

Enter choice (y/N):

Do you wish to use the same loginid and password?

Enter choice (y/N):

Connecting via Winsock to STN

LOGINID:

ssspt189dxw

STNLOGON timed out

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:sss189dxw

PASSWORD:

Green15

LOGINID/PASSWORD REJECTED

The loginid and/or password sent to STN were invalid.
You either typed them incorrectly, or line noise may
have corrupted them.

Do you wish to retry the logon?

Enter choice (y/N):

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspt189dxw

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * Welcome to STN International * * * * *

NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	JUL 28	CA/CAPLUS patent coverage enhanced
NEWS	3	JUL 28	EPFULL enhanced with additional legal status information from the epline Register
NEWS	4	JUL 28	IFICDB, IFIPAT, and IFIUDB reloaded with enhancements
NEWS	5	JUL 28	STN Viewer performance improved
NEWS	6	AUG 01	INPADOCDB and INPAFAMDB coverage enhanced
NEWS	7	AUG 13	CA/CAPLUS enhanced with printed Chemical Abstracts page images from 1967-1998
NEWS	8	AUG 15	CAOLD to be discontinued on December 31, 2008
NEWS	9	AUG 15	CAPLUS currency for Korean patents enhanced
NEWS	10	AUG 27	CAS definition of basic patents expanded to ensure comprehensive access to substance and sequence information
NEWS	11	SEP 18	Support for STN Express, Versions 6.01 and earlier, to be discontinued
NEWS	12	SEP 25	CA/CAPLUS current-awareness alert options enhanced to accommodate supplemental CAS indexing of exemplified prophetic substances
NEWS	13	SEP 26	WPIDS, WPINDEX, and WPIX coverage of Chinese and Korean patents enhanced
NEWS	14	SEP 29	IFICLS enhanced with new super search field
NEWS	15	SEP 29	EMBASE and EMBAL enhanced with new search and display fields
NEWS	16	SEP 30	CAS patent coverage enhanced to include exemplified prophetic substances identified in new Japanese-language patents
NEWS	17	OCT 07	EPFULL enhanced with full implementation of EPC2000
NEWS	18	OCT 07	Multiple databases enhanced for more flexible patent number searching
NEWS	19	OCT 22	Current-awareness alert (SDI) setup and editing enhanced
NEWS	20	OCT 22	WPIDS, WPINDEX, and WPIX enhanced with Canadian PCT Applications
NEWS	21	OCT 24	CHEMLIST enhanced with intermediate list of pre-registered REACH substances
NEWS EXPRESS	JUNE 27 08		CURRENT WINDOWS VERSION IS V8.3, AND CURRENT DISCOVER FILE IS DATED 23 JUNE 2008.
NEWS HOURS			STN Operating Hours Plus Help Desk Availability
NEWS LOGIN			Welcome Banner and News Items
NEWS IPC8			For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 18:42:33 ON 11 NOV 2008

=> index bioscience

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.21	0.21

FULL ESTIMATED COST

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 18:42:49 ON 11 NOV 2008

69 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view search error messages that display as 0* with SET DETAIL OFF.

=> s biliverdin and (ava or bird or avian or reptile or snake or reptilian) and reductase

4	FILE BIOSIS
1	FILE BIOTECHNO
10	FILE CAPLUS
2	FILE DGENE
1	FILE DRUGU
1	FILE EMBASE
2	FILE GENBANK
3	FILE IFIPAT
1	FILE MEDLINE
43 FILES SEARCHED...	
1	FILE TOXCENTER
75	FILE USPATFULL
8	FILE USPAT2
1	FILE WPIDS
1	FILE WPINDEX

14 FILES HAVE ONE OR MORE ANSWERS, 69 FILES SEARCHED IN STNINDEX

L1 QUE BILIVERDIN AND (AVA OR BIRD OR AVIAN OR REPTILE OR SNAKE OR REPTILIAN) AND REDUCTASE

=> s L1 and biliverdin reductase and (absorbence or absorbance)

2	FILE IFIPAT
55 FILES SEARCHED...	
14	FILE USPATFULL
1	FILE USPAT2
1	FILE WPIDS
1	FILE WPINDEX

5 FILES HAVE ONE OR MORE ANSWERS, 69 FILES SEARCHED IN STNINDEX

L2 QUE L1 AND BILIVERDIN REDUCTASE AND (ABSORBENCE OR ABSORBANCE)

=> file uspatfull uspat2

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	3.25	3.46

FILE 'USPATFULL' ENTERED AT 18:45:40 ON 11 NOV 2008
 CA INDEXING COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'USPAT2' ENTERED AT 18:45:40 ON 11 NOV 2008
 CA INDEXING COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)

```
=> s 12
L3          15 L2
```

```
=> rem dup 13
DUP IS NOT VALID HERE
```

The DELETE command is used to remove various items stored by the system.

To delete a saved query, saved answer set, saved L-number list, SDI request, batch request, mailing list, or user-defined cluster, format, or search field, enter the name. The name may include ? for left, right, or simultaneous left and right truncation.

Examples:

```
DELETE BIO?/Q      - delete query names starting with BIO
DELETE ?DRUG/A     - delete answer set names ending with DRUG
DELETE ?ELEC?/L    - delete L-number lists containing ELEC
DELETE ANTICOAG/S  - delete SDI request
DELETE ENZYME/B    - delete batch request
DELETE .MYCLUSTER  - delete user-defined cluster
DELETE .MYFORMAT   - delete user-defined display format
DELETE .MYFIELD    - delete user-defined search field
DELETE NAMELIST MYLIST - delete mailing list
```

To delete an ordered document or an offline print, enter its number.

Examples:

```
DELETE P123001C    - delete print request
DELETE D134002C    - delete document order request
```

To delete an individual L-number or range of L-numbers, enter the L-number or L-number range. You may also enter DELETE LAST followed by a number, n, to delete the last n L-numbers. RENUMBER or NORENUMBER may also be explicitly specified to override the value of SET RENUMBER.

Examples:

```
DELETE L21         - delete a single L-number
DELETE L3-L6       - delete a range of L-numbers
DELETE LAST 4      - delete the last 4 L-numbers
DELETE L33-        - delete L33 and any higher L-number
DELETE -L55        - delete L55 and any lower L-number
DELETE L2-L6 RENUMBER - delete a range of L-numbers and
                    renumber remaining L-numbers
DELETE RENUMBER    - renumber L-numbers after deletion of
                    intermediate L-numbers
```

Entire sets of saved items, SDI requests, batch requests, user-defined

items, or E-numbers can be deleted.

Examples:

```
DELETE SAVED/Q - delete all saved queries
DELETE SAVED/A - delete all saved answer sets
DELETE SAVED/L - delete all saved L-number lists
DELETE SAVED - delete all saved queries, answer sets,
               and L-number lists
DELETE SAVED/S - delete all SDI requests
DELETE SAVED/B - delete all batch requests
DELETE CLUSTER - delete all user-defined clusters
DELETE FORMAT - delete all user-defined display formats
DELETE FIELD - delete all user-defined search fields
DELETE SELECT - delete all E-numbers
DELETE HISTORY - delete all L-numbers and restart the
                 session at L1
```

To delete an entire multifile SDI request, enter DELETE and the name of the request. To delete a component from the multifile SDI, enter DELETE and the name of the component.

```
=> dup rem l3
PROCESSING COMPLETED FOR L3
L4          14 DUP REM L3 (1 DUPLICATE REMOVED)
```

```
=> d l4 1-14
```

```
L4  ANSWER 1 OF 14  USPATFULL on STN
AN  2008:253184  USPATFULL
TI  Advanced drug development and manufacturing
IN  Birnbaum, Eva R., Los Alamos, NM, UNITED STATES
    Koppisch, Andrew T., Flagstaff, AZ, UNITED STATES
    Baldwin, Sharon M., Santa Fe, NM, UNITED STATES
    Warner, Benjamin P., Los Alamos, NM, UNITED STATES
    McCleskey, T. Mark, Los Alamos, NM, UNITED STATES
    Stewart, Jeffrey Joseph, Los Alamos, NM, UNITED STATES
    Berger, Jennifer A., Los Alamos, NM, UNITED STATES
    Harris, Michael N., Los Alamos, NM, UNITED STATES
    Burrell, Anthony K., Los Alamos, NM, UNITED STATES
PI  US 20080220441      A1  20080911
AI  US 2007-974156      A1  20071010 (11)
RLI  Continuation-in-part of Ser. No. US 2001-859701, filed on 16 May 2001,
    PENDING Continuation-in-part of Ser. No. US 2002-206524, filed on 25 Jul
    2002, ABANDONED Continuation-in-part of Ser. No. US 2003-621825, filed
    on 16 Jul 2003, Pat. No. US 6858148
PRAI US 2006-850594P      20061010 (60)
DT  Utility
FS  APPLICATION
LN.CNT 10199
INCL  INCLM: 435/071.000
      INCLS: 436/501.000; 436/172.000; 436/086.000; 378/045.000
NCL  NCLM: 435/071.000
      NCLS: 436/501.000; 436/172.000; 436/086.000; 378/045.000
IC  IPCI  G01N0033-53 [I,A]; G01N0021-76 [I,A]; G01N0033-68 [I,A];
      G01N0023-223 [I,A]; G01N0023-22 [I,C*]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
```

```
L4  ANSWER 2 OF 14  USPATFULL on STN
AN  2007:177114  USPATFULL
TI  Genes associate with progression and response in chronic myeloid
    leukemia and uses thereof
```

IN Radich, Jerald P., Sammamish, WA, UNITED STATES
 Dai, Hongyue, Kenmore, WA, UNITED STATES
 Mao, Mao, Kirkland, WA, UNITED STATES
 Schelter, Janell M., Bellevue, WA, UNITED STATES
 Linsley, Peter S., Seattle, WA, UNITED STATES
 PI US 20070154931 A1 20070705
 AI US 2006-640517 A1 20061214 (11)
 PRAI US 2005-751455P 20051215 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 29037
 INCL INCLM: 435/006.000
 INCLS: 702/020.000
 NCL NCLM: 435/006.000
 NCLS: 702/020.000
 IC IPCI C12Q0001-68 [I,A]; G06F0019-00 [I,A]
 IPCR C12Q0001-68 [I,C]; C12Q0001-68 [I,A]; G06F0019-00 [I,C];
 G06F0019-00 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 3 OF 14 USPATFULL on STN
 AN 2006:294944 USPATFULL
 TI Assays for the detection of biliverdin in birds and reptiles
 IN Gregory, Christopher, 1015 Cooper Farm Road, Nicholson, GA, UNITED STATES 30565
 Ritchie, Branson W., Athens, GA, UNITED STATES
 PI US 20060252110 A1 20061109
 AI US 2003-525893 A1 20030827 (10)
 WO 2003-US27134 20030827
 20050708 PCT 371 date
 PRAI US 2002-406175P 20020827 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 1196
 INCL INCLM: 435/025.000
 NCL NCLM: 435/025.000
 IC IPCI C12Q0001-26 [I,A]
 IPCR C12Q0001-26 [I,C]; C12Q0001-26 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 4 OF 14 USPATFULL on STN
 AN 2006:131187 USPATFULL
 TI Red and near infrared fluorescent phytochrome
 IN Lagarias, John Clark, Davis, CA, UNITED STATES
 Fischer, Amanda J., Davis, CA, UNITED STATES
 PA The Regents of the University of California (U.S. corporation)
 PI US 20060110827 A1 20060525
 AI US 2005-123692 A1 20050505 (11)
 PRAI US 2004-569310P 20040506 (60)
 US 2004-598661P 20040803 (60)
 US 2004-640867P 20041230 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 5210
 INCL INCLM: 435/419.000
 INCLS: 536/023.600; 530/370.000; 435/006.000; 435/004.000
 NCL NCLM: 435/419.000
 NCLS: 435/004.000; 435/006.000; 530/370.000; 536/023.600
 IC IPCI C12Q0001-68 [I,A]; C12N0015-29 [I,A]
 IPCR C12Q0001-68 [I,A]; C12N0015-29 [I,C]; C12N0015-29 [I,A];
 C12Q0001-68 [I,C]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 5 OF 14 USPATFULL on STN
 AN 2006:60621 USPATFULL
 TI Genes and pathways differentially expressed in bipolar disorder and/or major depressive disorder
 IN Akil, Huda, Ann Arbor, MI, UNITED STATES
 Bunney, William E. JR., Laguna Beach, CA, UNITED STATES
 Choudary, Prabhakara V., Davis, CA, UNITED STATES
 Evans, Simon J., Milan, MI, UNITED STATES
 Jones, Edward G., Winters, CA, UNITED STATES
 Li, Jun, Palo Alto, CA, UNITED STATES
 Lopez, Juan F., Ann Arbor, MI, UNITED STATES
 Lyons, David M., Palo Alto, CA, UNITED STATES
 Molnar, Margherita, Davis, CA, UNITED STATES
 Myers, Richard M., Stanford, CA, UNITED STATES
 Schatzberg, Alan F., Los Altos, CA, UNITED STATES
 Stein, Richard, Irvine, CA, UNITED STATES
 Thompson, Robert C., Ann Arbor, MI, UNITED STATES
 Tomita, Hiroaki, Irvine, CA, UNITED STATES
 Vawter, Marquis P., Laguna Niguel, CA, UNITED STATES
 Watson, Stanley J., Ann Arbor, MI, UNITED STATES
 PA The Board of Trustees of the Leland Stanford Junior University of Stanford, Palo Alto, CA, UNITED STATES (U.S. corporation)
 PI US 20060051786 A1 20060309
 AI US 2005-158530 A1 20050621 (11)
 PRAI US 2004-581998P 20040621 (60)
 US 2004-621252P 20041022 (60)
 US 2005-667296P 20050331 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 8628
 INCL INCLM: 435/006.000
 INCLS: 435/007.100
 NCL NCLM: 435/006.000
 NCLS: 435/007.100
 IC IPCI C12Q0001-68 [I,A]; G01N0033-53 [I,A]
 IPCR C12Q0001-68 [I,A]; C12Q0001-68 [I,C]; G01N0033-53 [I,C];
 G01N0033-53 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 6 OF 14 USPATFULL on STN
 AN 2005:241176 USPATFULL
 TI Compositions and methods for diagnosing and treating mental disorders
 IN Akil, Huda, Ann Arbor, MI, UNITED STATES
 Atz, Mary, Tustin, CA, UNITED STATES
 Bunney, William E. JR., Laguna Beach, CA, UNITED STATES
 Choudary, Prabhakara V., Davis, CA, UNITED STATES
 Evans, Simon J., Milan, MI, UNITED STATES
 Jones, Edward G., Winters, CA, UNITED STATES
 Li, Jun, Palo Alto, CA, UNITED STATES
 Lopez, Juan F., Ann Arbor, MI, UNITED STATES
 Myers, Richard M., Stanford, CA, UNITED STATES
 Thompson, Robert C., Ann Arbor, MI, UNITED STATES
 Tomita, Hiroaki, Irvine, CA, UNITED STATES
 Vawter, Marquis P., Niguel, CA, UNITED STATES
 Watson, Stanley, Ann Arbor, MI, UNITED STATES
 PI US 20050209181 A1 20050922
 AI US 2004-982556 A1 20041104 (10)
 PRAI US 2003-517751P 20031105 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 11427

INCL INCLM: 514/044.000
 INCLS: 435/006.000; 514/220.000; 514/259.410; 514/469.000
 NCL NCLM: 514/044.000
 NCLS: 435/006.000; 514/220.000; 514/259.410; 514/469.000
 IC [7]
 ICM C12Q001-68
 ICS A61K048-00; A61K031-519
 IPCI C12Q0001-68 [ICM,7]; A61K0048-00 [ICS,7]; A61K0031-519 [ICS,7]
 IPCR A61B [I,S]; A61K0031-519 [I,C*]; A61K0031-519 [I,A]; A61K0048-00 [I,C*]; A61K0048-00 [I,A]; C12Q0001-68 [I,C*]; C12Q0001-68 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 7 OF 14 USPATFULL on STN
 AN 2004:38576 USPATFULL
 TI Methods of diagnosis of breast cancer, compositions and methods of screening for modulators of breast cancer
 IN Mack, David H., Menlo Park, CA, UNITED STATES
 Gish, Kurt C., San Francisco, CA, UNITED STATES
 Afar, Daniel, Brisbane, CA, UNITED STATES
 PA Eos Technology, Inc., South San Francisco, CA, UNITED STATES, 94080-7019 (U.S. corporation)
 PI US 20040029114 A1 20040212
 AI US 2002-58270 A1 20020124 (10)
 PRAI US 2001-263965P 20010124 (60)
 US 2001-265928P 20010202 (60)
 US 2001-282698P 20010409 (60)
 US 2001-288590P 20010504 (60)
 US 2001-294443P 20010529 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 42494
 INCL INCLM: 435/006.000
 INCLS: 435/069.100; 435/320.100; 435/325.000; 530/350.000; 536/023.500
 NCL NCLM: 435/006.000
 NCLS: 435/069.100; 435/320.100; 435/325.000; 530/350.000; 536/023.500
 IC [7]
 ICM C12Q001-68
 ICS C07H021-04; C07K014-72; C12P021-02; C12N005-06
 IPCI C12Q0001-68 [ICM,7]; C07H0021-04 [ICS,7]; C07H0021-00 [ICS,7,C*]; C07K0014-72 [ICS,7]; C07K0014-435 [ICS,7,C*]; C12P0021-02 [ICS,7]; C12N0005-06 [ICS,7]
 IPCR C07K0014-435 [I,C*]; C07K0014-47 [I,A]; C12Q0001-68 [I,C*]; C12Q0001-68 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 8 OF 14 USPATFULL on STN
 AN 2003:324595 USPATFULL
 TI Methods of diagnosis of Hepatitis C infection, compositions and methods of screening for modulators of Hepatitis C infection
 IN Yat Wah Tom, Edward, Sacramento, CA, UNITED STATES
 Zlotnik, Albert, Palo Alto, CA, UNITED STATES
 PA Eos Biotechnology, Inc., South San Francisco, CA (U.S. corporation)
 PI US 20030228570 A1 20031211
 AI US 2003-366435 A1 20030212 (10)
 RLI Continuation of Ser. No. US 2002-206473, filed on 24 Jul 2002, ABANDONED
 PRAI US 2002-366782P 20020321 (60)
 US 2001-308188P 20010726 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 22742
 INCL INCLM: 435/005.000
 INCLS: 435/006.000; 435/069.300; 435/320.100; 435/325.000; 530/350.000;

530/388.300; 536/023.720
NCL NCLM: 435/005.000
NCLS: 435/006.000; 435/069.300; 435/320.100; 435/325.000; 530/350.000;
530/388.300; 536/023.720
IC [7]
ICM C12Q001-70
ICS C12Q001-68; C07H021-04; C07K014-02; C07K016-08; C12P021-02;
C12N005-06
IPCI C12Q0001-70 [ICM,7]; C12Q0001-68 [ICS,7]; C07H0021-04 [ICS,7];
C07H0021-00 [ICS,7,C*]; C07K0014-02 [ICS,7]; C07K0014-005
[ICS,7,C*]; C07K0016-08 [ICS,7]; C12P0021-02 [ICS,7]; C12N0005-06
[ICS,7]
IPCR C12Q0001-70 [I,C*]; C12Q0001-70 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 9 OF 14 USPATFULL on STN
AN 2003:220740 USPATFULL
TI Methods and compositions for diagnosing and treating rheumatoid
arthritis
IN Pittman, Debra D., Windham, NH, UNITED STATES
Feldman, Jeffrey L., Arlington, MA, UNITED STATES
Shields, Kathleen M., Harvard, MA, UNITED STATES
Trepicchio, William L., Andover, MA, UNITED STATES
PI US 20030154032 A1 20030814
AI US 2001-23451 A1 20011217 (10)
PRAI US 2000-255861P 20001215 (60)
DT Utility
FS APPLICATION
LN.CNT 25385
INCL INCLM: 702/020.000
NCL NCLM: 702/020.000
IC [7]
ICM G06F019-00
ICS G01N033-48
IPCI G06F0019-00 [ICM,7]; G01N0033-48 [ICS,7]
IPCR A61K0038-00 [N,C*]; A61K0038-00 [N,A]; C07K0014-435 [I,C*];
C07K0014-47 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 10 OF 14 USPATFULL on STN
AN 2003:120026 USPATFULL
TI Identification of modulatory molecules using inducible promoters
IN Brown, Steven J., San Diego, CA, UNITED STATES
Dunnington, Damien J., San Diego, CA, UNITED STATES
Clark, Imran, San Diego, CA, UNITED STATES
PI US 20030082511 A1 20030501
AI US 2001-965201 A1 20010925 (9)
DT Utility
FS APPLICATION
LN.CNT 5526
INCL INCLM: 435/004.000
INCLS: 435/006.000
NCL NCLM: 435/004.000
NCLS: 435/006.000
IC [7]
ICM C12Q001-00
ICS C12Q001-68
IPCI C12Q0001-00 [ICM,7]; C12Q0001-68 [ICS,7]
IPCR G01N0033-50 [I,C*]; G01N0033-50 [I,A]; G01N0033-68 [I,C*];
G01N0033-68 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 11 OF 14 USPATFULL on STN DUPLICATE 1
 AN 2002:301655 USPATFULL
 TI Compounds and methods for regulating cell differentiation
 IN Falchuk, Kenneth H., Newton, MA, UNITED STATES
 PA President & Fellows of Harvard College, Cambridge, MA, UNITED STATES
 (U.S. corporation)
 PI US 20020169201 A1 20021114
 US 6902881 B2 20050607
 AI US 2001-8356 A1 20011113 (10)
 RLI Continuation-in-part of Ser. No. US 2001-977866, filed on 15 Oct 2001,
 PENDING
 PRAI US 2000-240497P 20001013 (60)
 US 2000-247299P 20001110 (60)
 US 2001-262233P 20010117 (60)
 US 2001-264814P 20010129 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 4893
 INCL INCLM: 514/422.000
 INCLS: 548/518.000
 NCL NCLM: 435/001.100; 514/422.000
 NCLS: 435/325.000; 514/359.000; 514/422.000; 548/518.000
 IC [7]
 ICM A61K031-4025
 ICS C07D043-14
 IPCI A61K0031-4025 [ICM,7]; C07D0043-14 [ICS,7]
 IPCI-2 A01N0001-00 [ICM,7]; A01N0043-38 [ICS,7]; A01N0043-34 [ICS,7,C*];
 C12N0005-02 [ICS,7]; A61K0031-409 [ICS,7]
 IPCR A61K0031-409 [I,C*]; A61K0031-409 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 12 OF 14 USPATFULL on STN
 AN 97:68351 USPATFULL
 TI Nucleic acid preparation methods
 IN Lin, Lily, Berkeley, CA, United States
 PA HRI Research, Inc., Concord, CA, United States (U.S. corporation)
 PI US 5654179 19970805
 AI US 1994-317220 19941003 (8)
 RLI Continuation of Ser. No. US 1993-44649, filed on 8 Apr 1993, now
 abandoned which is a continuation-in-part of Ser. No. US 1992-901545,
 filed on 19 Jun 1992, now abandoned which is a continuation-in-part of
 Ser. No. US 1990-614921, filed on 14 Nov 1990, now patented, Pat. No. US
 5284940, issued on 8 Feb 1994
 DT Utility
 FS Granted
 LN.CNT 2765
 INCL INCLM: 435/091.200
 INCLS: 435/270.000; 436/177.000; 436/825.000; 536/025.400; 536/025.410;
 536/025.420
 NCL NCLM: 435/091.200
 NCLS: 435/270.000; 436/177.000; 436/825.000; 536/025.400; 536/025.410;
 536/025.420
 IC [6]
 ICM C12P019-34
 ICS C07H021-02
 IPCI C12P0019-34 [ICM,6]; C12P0019-00 [ICM,6,C*]; C07H0021-02 [ICS,6];
 C07H0021-00 [ICS,6,C*]
 IPCR C12N0015-10 [I,C*]; C12N0015-10 [I,A]; C12Q0001-68 [I,C*];
 C12Q0001-68 [I,A]; C12Q0001-70 [I,C*]; C12Q0001-70 [I,A]
 EXF 435/91.2; 435/270; 536/25.4; 536/25.41; 536/25.42; 436/177; 436/825
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 13 OF 14 USPATFULL on STN
 AN 97:31574 USPATFULL
 TI Nucleic acid preparation methods
 IN Lin, Lily, Berkeley, CA, United States
 Cimino, George, Richmond, CA, United States
 Zhu, Yu S., Richmond, CA, United States
 PA HRI Research, Inc., Concord, CA, United States (U.S. corporation)
 PI US 5620852 19970415
 AI US 1994-332616 19941031 (8)
 RLI Continuation of Ser. No. US 1992-901545, filed on 19 Jun 1992, now
 abandoned which is a continuation-in-part of Ser. No. US 1990-614921,
 filed on 14 Nov 1990, now patented, Pat. No. US 5284940
 DT Utility
 FS Granted
 LN.CNT 2451
 INCL INCLM: 435/006.000
 INCLS: 536/025.300; 536/022.100; 435/091.100
 NCL NCLM: 435/006.000
 NCLS: 435/091.100; 536/022.100; 536/025.300
 IC [6]
 ICM C12Q001-68
 ICS C12P019-34
 IPCI C12Q0001-68 [ICM,6]; C12P0019-34 [ICS,6]; C12P0019-00 [ICS,6,C*]
 IPCR C12N0015-10 [I,C*]; C12N0015-10 [I,A]; C12Q0001-68 [I,C*];
 C12Q0001-68 [I,A]; C12Q0001-70 [I,C*]; C12Q0001-70 [I,A]
 EXF 536/25.4; 536/25.41; 536/25.42; 536/25.3; 435/6
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 14 OF 14 USPATFULL on STN
 AN 94:11507 USPATFULL
 TI Preparation for nucleic acid samples
 IN Lin, Lily, Berkeley, CA, United States
 Isaacs, Stephen T., Orinda, CA, United States
 Hearst, John E., Berkeley, CA, United States
 PA HRI Research, Inc., Concord, CA, United States (U.S. corporation)
 PI US 5284940 19940208
 AI US 1990-614921 19901114 (7)
 DT Utility
 FS Granted
 LN.CNT 2082
 INCL INCLM: 536/025.400
 INCLS: 536/025.410; 536/025.420; 435/006.000; 435/270.000
 NCL NCLM: 536/025.400
 NCLS: 435/006.000; 435/270.000; 536/025.410; 536/025.420
 IC [5]
 ICM C07H023-00
 ICS C12Q001-68; C12N001-08
 IPCI C07H0023-00 [ICM,5]; C12Q0001-68 [ICS,5]; C12N0001-08 [ICS,5]
 IPCR C07K0016-44 [I,C*]; C07K0016-44 [I,A]; C12N0015-10 [I,C*];
 C12N0015-10 [I,A]; C12Q0001-68 [I,C*]; C12Q0001-68 [I,A];
 C12Q0001-70 [I,C*]; C12Q0001-70 [I,A]
 EXF 435/270; 435/280; 435/6; 435/262; 435/259; 435/805; 536/27; 536/28;
 536/25.4; 536/25.41; 536/25.42
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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=> logoff

ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF

LOGOFF? (Y)/N/HOLD:y

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

65.70

69.16

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NEWS	2	DEC 01	ChemPort single article sales feature unavailable
NEWS	3	FEB 02	Simultaneous left and right truncation (SLART) added for CERAB, COMPUAB, ELCOM, and SOLIDSTATE
NEWS	4	FEB 02	GENBANK enhanced with SET PLURALS and SET SPELLING
NEWS	5	FEB 06	Patent sequence location (PSL) data added to USGENE
NEWS	6	FEB 10	COMPENDEX reloaded and enhanced
NEWS	7	FEB 11	WTEXTILES reloaded and enhanced
NEWS	8	FEB 19	New patent-examiner citations in 300,000 CA/CAPLUS patent records provide insights into related prior art
NEWS	9	FEB 19	Increase the precision of your patent queries -- use terms from the IPC Thesaurus, Version 2009.01
NEWS	10	FEB 23	Several formats for image display and print options discontinued in USPATFULL and USPAT2
NEWS	11	FEB 23	MEDLINE now offers more precise author group fields and 2009 MeSH terms
NEWS	12	FEB 23	TOXCENTER updates mirror those of MEDLINE - more precise author group fields and 2009 MeSH terms
NEWS	13	FEB 23	Three million new patent records blast AEROSPACE into STN patent clusters
NEWS	14	FEB 25	USGENE enhanced with patent family and legal status display data from INPADOCDB
NEWS	15	MAR 06	INPADOCDB and INPAFAMDB enhanced with new display formats
NEWS	16	MAR 11	EPFULL backfile enhanced with additional full-text applications and grants
NEWS	17	MAR 11	ESBIOBASE reloaded and enhanced
NEWS	18	MAR 20	CAS databases on STN enhanced with new super role for nanomaterial substances
NEWS	19	MAR 23	CA/CAPLUS enhanced with more than 250,000 patent equivalents from China
NEWS	20	MAR 30	IMSPATENTS reloaded and enhanced
NEWS	21	APR 03	CAS coverage of exemplified prophetic substances enhanced
NEWS	22	APR 07	STN is raising the limits on saved answers
NEWS	23	APR 24	CA/CAPLUS now has more comprehensive patent assignee information
NEWS	24	APR 26	USPATFULL and USPAT2 enhanced with patent assignment/reassignment information
NEWS	25	APR 28	CAS patent authority coverage expanded

NEWS 26 APR 28 ENCOMPLIT/ENCOMPLIT2 search fields enhanced
 NEWS 27 APR 28 Limits doubled for structure searching in CAS
 REGISTRY
 NEWS 28 MAY 08 STN Express, Version 8.4, now available
 NEWS 29 MAY 11 STN on the Web enhanced
 NEWS 30 MAY 11 BEILSTEIN substance information now available on
 STN Easy
 NEWS 31 MAY 14 DGENE, PCTGEN and USGENE enhanced with increased
 limits for exact sequence match searches and
 introduction of free HIT display format
 NEWS 32 MAY 15 INPADOCDB and INPAFAMDB enhanced with Chinese legal
 status data

NEWS EXPRESS JUNE 27 08 CURRENT WINDOWS VERSION IS V8.3,
 AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2009.

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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 00:15:26 ON 24 MAY 2009

=> index bioscience

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.22	0.22

FULL ESTIMATED COST

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
 AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
 CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
 DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 00:15:56 ON 24 MAY 2009

68 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
 search error messages that display as 0* with SET DETAIL OFF.

=> s bilverdin and measur?(p)absorbance and (bird or avian or reptil?)

0* FILE ADISNEWS
 0* FILE ANTE
 0* FILE AQUALINE
 0* FILE BIOENG
 0* FILE BIOTECHABS
 0* FILE BIOTECHDS
 0* FILE BIOTECHNO
 0* FILE CEABA-VTB

16 FILES SEARCHED...

0* FILE CIN

27 FILES SEARCHED...

0* FILE FOMAD

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0* FILE FOREGE
0* FILE FROSTI
0* FILE FSTA
0* FILE KOSMET
0* FILE NTIS
44 FILES SEARCHED...
0* FILE NUTRACEUT
0* FILE PASCAL
0* FILE PHARMAML
58 FILES SEARCHED...
1 FILE USPATFULL
0* FILE WATER

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1 FILES HAVE ONE OR MORE ANSWERS, 68 FILES SEARCHED IN STNINDEX

L1 QUE BILVERDIN AND MEASUR?(P) ABSORBANCE AND (BIRD OR AVIAN OR REPTIL?)

=> file uspatfull

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.72	2.94

FILE 'USPATFULL' ENTERED AT 00:18:18 ON 24 MAY 2009
CA INDEXING COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 21 May 2009 (20090521/PD)
FILE LAST UPDATED: 21 May 2009 (20090521/ED)
HIGHEST GRANTED PATENT NUMBER: US7536727
HIGHEST APPLICATION PUBLICATION NUMBER: US20090133177
CA INDEXING IS CURRENT THROUGH 21 May 2009 (20090521/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 21 May 2009 (20090521/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2009

USPATFULL now includes complete International Patent Classification (IPC) reclassification data for the third quarter of 2008.

=> s l1

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3 BILVERDIN
2008607 MEASUR?
96024 ABSORBANCE
50455 MEASUR?(P) ABSORBANCE
35514 BIRD
22835 AVIAN
8248 REPTIL?

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L2 1 BILVERDIN AND MEASUR?(P) ABSORBANCE AND (BIRD OR AVIAN OR REPTIL ?)

=> d l2

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L2 ANSWER 1 OF 1 USPATFULL on STN
AN 2003:220740 USPATFULL
TI Methods and compositions for diagnosing and treating rheumatoid
arthritis
IN Pittman, Debra D., Windham, NH, UNITED STATES
Feldman, Jeffrey L., Arlington, MA, UNITED STATES
Shields, Kathleen M., Harvard, MA, UNITED STATES
Trepicchio, William L., Andover, MA, UNITED STATES
PI US 20030154032 A1 20030814
AI US 2001-23451 A1 20011217 (10)
PRAI US 2000-255861P 20001215 (60)
DT Utility

```

```

FS      APPLICATION
LN.CNT  25385
INCL    INCLM: 702/020.000
NCL     NCLM: 702/020.000
IC      [7]
        ICM      G06F019-00
        ICS      G01N033-48
        IPCI     G06F0019-00 [ICM,7]; G01N0033-48 [ICS,7]
        IPCR     A61K0038-00 [N,C*]; A61K0038-00 [N,A]; C07K0014-435 [I,C*];
                C07K0014-47 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

```

=> d kwic 12

```

L2      ANSWER 1 OF 1  USPATFULL on STN
DETD    . . . for 16 hours. Labeled RNA was purified with the use of an
        RNeasy (Qiagen). The RNA yield was quantitated by measuring
        absorbance at 260 nm.
DETD    . . . 19q13.3          ribosomal protein S11
        ribosomal protein S11
JUN      U65928_at          U65928          PASS      8      8.75      PASS
        11      8      6.55      1.34      1.34      JUN      1p32-p31
        Avian sarcoma virus 17      v-jun avian sarcoma virus
        (v-jun) oncogene homolog      17 oncogene homolog
INT6      U62962_at          U62962          PASS      9      52.67      PASS
        13      9      39.46      1.33      1.33      EIF3S6. . .
DETD      . . . protein 2, 56kD,
        DOK2
37002_at      BLVRB          D32143          6      Pass      25 00      11 47
        13      Pass      TRUE      FALSE      FALSE      11 85      2.11
        bilverdin reductase B          19q13 1-
        (flavin reductase          q13 2
        (NADPH), BLVRB
1357_at      USP4          U20657          6      Pass      12 33      4.59
        13      Pass      TRUE. . .
DETD      . . . protein 1,          6p21.3
        KING1
1295_at      RELA          L19067          6      Pass      39.17      15 38
        13      Pass      TRUE      FALSE      FALSE      19 46      2.01
        v-rel avian          11q13
        reticuloendotheliosis          viral
        oncogene homolog A
        (nuclear factor of kappa
        light polypepode gene
        enhancer in B-cells 3
        (p65)), RELA
32271. . .
DETD      . . . 19
        complement (adipsin), DF

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36645_at	RELA	L19067	6	Pass	33 67	12.82
13	Pass	TRUE	FALSE	FALSE	16 85	2.00
v-rel avian 11q13						

reticuloendotheliosis viral

oncogene homolog A

(nuclear factor af kappa

light polypeptide gene

enhancer in B-cells 3

(p65)), RELA

41471_at SI00A9. . .

DETD

carrier protein, 17

. . .

E2-EPF

570_at	RELB	M83221	6	Pass	8.00	3 35
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6	Fail	FALSE	TRUE	FALSE	3 67	2.18
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v-tel avian

reticuloendotheliosis viral

oncogene homolog B

(nuclear factor of kappa

DETD . . . including both growth

and

(cytokine suppression

responsive apoptosis.

protein cr6).

S74567	1.00	-1.41	3.30	0.41	2.84	1.39	2.88	0.80	2.39
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1.21	avian	S74567	transcription	the
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c-maf interaction 8 61.0 cM

musculoaponeu-

factor maf2 site was

mapped to the

rotic fibrosarcoma

(proto-oncogene sequence.

. .

=> d hist

(FILE 'HOME' ENTERED AT 00:15:26 ON 24 MAY 2009)

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 00:15:56 ON 24 MAY 2009
SEA BILVERDIN AND MEASUR?(P)ABSORBANCE AND (BIRD OR AVIAN OR RE

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0* FILE BIOENG
0* FILE BIOTECHABS
0* FILE BIOTECHDS
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0* FILE CEABA-VTB
0* FILE CIN

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0* FILE NUTRACEUT
0* FILE PASCAL
0* FILE PHARMAML
1 FILE USPATFULL
0* FILE WATER

L1 QUE BILVERDIN AND MEASUR?(P) ABSORBANCE AND (BIRD OR AVIAN OR R

FILE 'USPATFULL' ENTERED AT 00:18:18 ON 24 MAY 2009

L2 1 S L1

=> d 12 1

L2 ANSWER 1 OF 1 USPATFULL on STN

AN 2003:220740 USPATFULL

TI Methods and compositions for diagnosing and treating rheumatoid
arthritis

IN Pittman, Debra D., Windham, NH, UNITED STATES
Feldman, Jeffrey L., Arlington, MA, UNITED STATES
Shields, Kathleen M., Harvard, MA, UNITED STATES
Trepicchio, William L., Andover, MA, UNITED STATES

PI US 20030154032 A1 20030814

AI US 2001-23451 A1 20011217 (10)

PRAI US 2000-255861P 20001215 (60)

DT Utility

FS APPLICATION

LN.CNT 25385

INCL INCLM: 702/020.000

NCL NCLM: 702/020.000

IC [7]

ICM G06F019-00

ICS G01N033-48

IPCI G06F0019-00 [ICM,7]; G01N0033-48 [ICS,7]

IPCR A61K0038-00 [N,C*]; A61K0038-00 [N,A]; C07K0014-435 [I,C*];
C07K0014-47 [I,A]

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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11.26

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NEWS 3 JAN 25 Annual Reload of MEDLINE database
NEWS 4 FEB 16 STN Express Maintenance Release, Version 8.4.2, Is
Now Available for Download
NEWS 5 FEB 16 Derwent World Patents Index (DWPI) Revises Indexing
of Author Abstracts
NEWS 6 FEB 16 New FASTA Display Formats Added to USGENE and PCTGEN
NEWS 7 FEB 16 INPADOCDB and INPAFAMDB Enriched with New Content
and Features
NEWS 8 FEB 16 INSPEC Adding Its Own IPC codes and Author's E-mail
Addresses
NEWS 9 APR 02 CAS Registry Number Crossover Limits Increased to
500,000 in Key STN Databases
NEWS 10 APR 02 PATDPAFULL: Application and priority number formats
enhanced
NEWS 11 APR 02 DWPI: New display format ALLSTR available
NEWS 12 APR 02 New Thesaurus Added to Derwent Databases for Smooth
Sailing through U.S. Patent Codes
NEWS 13 APR 02 EMBASE Adds Unique Records from MEDLINE, Expanding
Coverage back to 1948
NEWS 14 APR 07 CA/CAPLUS CLASS Display Streamlined with Removal of
Pre-IPC 8 Data Fields
NEWS 15 APR 07 50,000 World Traditional Medicine (WTM) Patents Now
Available in CAPLUS
NEWS 16 APR 07 MEDLINE Coverage Is Extended Back to 1947

NEWS EXPRESS FEBRUARY 15 10 CURRENT WINDOWS VERSION IS V8.4.2,
AND CURRENT DISCOVER FILE IS DATED 15 JANUARY 2010.

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=> index bioscience

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SINCE FILE	TOTAL
ENTRY	SESSION

FULL ESTIMATED COST

0.22	0.22
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AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 19:54:11 ON 29 APR 2010

63 FILES IN THE FILE LIST IN STNINDEX

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search error messages that display as 0* with SET DETAIL OFF.

=> s biliverdin and (avian or bird or reptil?) and (liver or hepatic) and function

1 FILE CABA
1 FILE CAPLUS
23 FILES SEARCHED...
1 FILE EMBASE
2 FILE IFIPAT
1 FILE MEDLINE
79 FILE USPATFULL
13 FILE USPAT2

61 FILES SEARCHED...

7 FILES HAVE ONE OR MORE ANSWERS, 63 FILES SEARCHED IN STNINDEX

L1 QUE BILIVERDIN AND (AVIAN OR BIRD OR REPTIL?) AND (LIVER OR HEPATIC) AND F
UNCTION

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FULL ESTIMATED COST	4.83	5.05

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FILE 'USPATFULL' ENTERED AT 19:58:09 ON 29 APR 2010
CA INDEXING COPYRIGHT (C) 2010 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'USPAT2' ENTERED AT 19:58:09 ON 29 APR 2010
CA INDEXING COPYRIGHT (C) 2010 AMERICAN CHEMICAL SOCIETY (ACS)

=> s l1
L2 98 L1

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 96 DUP REM L2 (2 DUPLICATES REMOVED)

=> s l3 and (hepatic or liver)(p)functi?
L4 53 L3 AND (HEPATIC OR LIVER) (P) FUNCTI?

=> s L4 and absorbance
L5 24 L4 AND ABSORBANCE

=> s L5 and reductase
L6 19 L5 AND REDUCTASE

=> d 16 1-19

L6 ANSWER 1 OF 19 IFIPAT COPYRIGHT 2010 IFI on STN
AN 11880348 IFIPAT;IFIUDB;IFICDB
TI Advanced drug development and manufacturing; Using x-ray fluorescence to
monitor protein ligand binding; rational drug design and screening
IN Baldwin Sharon M; Berger Jennifer A; Birnbaum Eva R; Burrell Anthony K;
Harris Michael N; Koppisch Andrew T; McCleskey T Mark; Stewart Jeffrey
Joseph; Warner Benjamin P
PA Unassigned Or Assigned To Individual (68000)
PPA LOS ALAMOS NATIONAL SECURITY LLC (Probable)
PI US 20080220441 A1 20080911
AI US 2007-974156 20071010 (11)
RLI US 2001-859701 20010516 CONTINUATION-IN-PART PENDING
US 2002-206524 20020725 CONTINUATION-IN-PART ABANDONED
US 2003-621825 20030716 CONTINUATION-IN-PART 6858148
PRAI US 2006-850594P 20061010 (Provisional)
FI US 20080220441 20080911
US 6858148
DT Utility; Patent Application - First Publication
FS CHEMICAL
APPLICATION
OS CA 149:326754
ED Entered STN: 17 Sep 2008
Last Updated on STN: 16 Mar 2009
CLMN 48

L6 ANSWER 2 OF 19 USPATFULL on STN
AN 2010:97564 USPATFULL
TI Identification, Monitoring and Treatment of Disease and Characterization
of Biological Condition Using Gene Expression Profiles
IN Bevilacqua, Michael P., Boulder, CO, UNITED STATES
Cheronis, John C., Conifer, CO, UNITED STATES
Tryon, Victor, Loveland, CO, UNITED STATES
Bankaitis-Davis, Danute M., Longmont, CO, UNITED STATES
PA Source Precision Medicine, Inc., Boulder, CO, UNITED STATES (U.S.
corporation)
PI US 20100086935 A1 20100408
AI US 2009-609578 A1 20091030 (12)
RLI Continuation of Ser. No. US 2005-158504, filed on 22 Jun 2005, ABANDONED
Continuation of Ser. No. US 2002-291856, filed on 8 Nov 2002, Pat. No.
US 6964850
PRAI US 2001-348213P 20011109 (60)
US 2001-340881P 20011207 (60)
US 2002-369633P 20020403 (60)
US 2002-376997P 20020430 (60)
DT Utility
FS APPLICATION
LN.CNT 4634
INCL INCLM: 435 6
INCLS: 702/020.000
NCL NCLM: 435 6
NCLS: 702/020.000
IC IPCI C12Q0001-68 [I,A]; G01N0033-48 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 3 OF 19 USPATFULL on STN
 AN 2009:333144 USPATFULL
 TI METHODS FOR DIAGNOSING AND MONITORING THE STATUS OF SYSTEMIC LUPUS
 ERYTHEMATOSUS
 IN LAL, Preeti G., Santa Clara, CA, UNITED STATES
 Williams, Gavin E., Menlo Park, CA, UNITED STATES
 Fry, Kirk E., Palo Alto, CA, UNITED STATES
 Sun, Jingtao, Foster City, CA, UNITED STATES
 Dedrick, Russell L., Kensington, CA, UNITED STATES
 PA XDX, Inc., Brisbane, CA, UNITED STATES (U.S. corporation)
 PI US 20090298060 A1 20091203
 AI US 2007-938227 A1 20071109 (11)
 PRAI US 2006-858147P 20061109 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 9197
 INCL INCLM: 435 6
 NCL NCLM: 435/006.000
 IC IPCI C12Q0001-68 [I,A]
 IPCR C12Q0001-68 [I,C]; C12Q0001-68 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 4 OF 19 USPATFULL on STN
 AN 2009:232884 USPATFULL
 TI Rationale, Methods, and Assays for Identifying Human and Non-Human
 Primate Taste Specific Genes and Use Thereof in Taste Modulator and
 Therapeutic Screening Assays
 IN Moyer, Bryan, San Diego, CA, UNITED STATES
 Zlotnik, Albert, San Diego, CA, UNITED STATES
 Hevezi, Peter, Encinitas, CA, UNITED STATES
 Soto, Hortensia, San Diego, CA, UNITED STATES
 Kalabat, Dalia, El Cajon, CA, UNITED STATES
 Lu, Min, San Diego, CA, UNITED STATES
 Gao, Na, San Diego, CA, UNITED STATES
 White, Evan Carl, Fair Oaks, CA, UNITED STATES
 PI US 20090208946 A1 20090820
 AI US 2008-134302 A1 20080606 (12)
 PRAI US 2007-929017P 20070608 (60)
 US 2007-929007P 20070608 (60)
 US 2007-947052P 20070629 (60)
 US 2007-935297P 20070803 (60)
 US 2007-987611P 20071113 (60)
 US 2007-988938P 20071119 (60)
 US 2007-991274P 20071130 (60)
 US 2007-991289P 20071130 (60)
 US 2007-992502P 20071205 (60)
 US 2007-992517P 20071205 (60)
 US 2007-17244P 20071228 (61)
 US 2008-21437P 20080116 (61)
 US 2008-43257P 20080408 (61)
 US 2008-53310P 20080515 (61)
 DT Utility
 FS APPLICATION
 LN.CNT 24869
 INCL INCLM: 435 6
 INCLS: 435/029.000; 435/366.000; 435/363.000
 NCL NCLM: 435/006.000
 NCLS: 435/029.000; 435/363.000; 435/366.000
 IC IPCI C12Q0001-68 [I,A]; C12Q0001-02 [I,A]; C12N0005-08 [I,A]
 IPCR C12Q0001-68 [I,C]; C12Q0001-68 [I,A]; C12N0005-08 [I,C];
 C12N0005-08 [I,A]; C12Q0001-02 [I,C]; C12Q0001-02 [I,A]

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 5 OF 19 USPATFULL on STN
AN 2009:11620 USPATFULL
TI Materials and Methods for Diagnosis and Treatment of Chronic Fatigue Syndrome
IN Gow, John, Glasgow, UNITED KINGDOM
Chaudhuri, Abhijit, Glasgow, UNITED KINGDOM
PI US 20090010908 A1 20090108
AI US 2006-815290 A1 20060201 (11)
WO 2006-GB332 20060201
20080716 PCT 371 date
PRAI GB 2005-2042 20050201
DT Utility
FS APPLICATION
LN.CNT 11046
INCL INCLM: 424/094.100
INCLS: 506/023.000; 506/024.000; 506/026.000; 506/013.000; 514/154.000;
514/406.000; 514/152.000; 514/179.000; 435 6
NCL NCLM: 424/094.100
NCLS: 435/006.000; 506/013.000; 506/023.000; 506/024.000; 506/026.000;
514/152.000; 514/154.000; 514/179.000; 514/406.000
IC IPCI A61K0031-122 [I,A]; C40B0050-00 [I,A]; C40B0050-02 [I,A];
C40B0050-06 [I,A]; A61K0031-573 [I,A]; A61K0031-57 [I,C*];
C12Q0001-68 [I,A]; A61K0031-415 [I,A]; C40B0040-00 [I,A];
A61K0031-65 [I,A]
IPCR A61K0031-122 [I,C]; A61K0031-122 [I,A]; A61K0031-415 [I,C];
A61K0031-415 [I,A]; A61K0031-57 [I,C]; A61K0031-573 [I,A];
A61K0031-65 [I,C]; A61K0031-65 [I,A]; C12Q0001-68 [I,C];
C12Q0001-68 [I,A]; C40B0040-00 [I,C]; C40B0040-00 [I,A];
C40B0050-00 [I,C]; C40B0050-00 [I,A]; C40B0050-02 [I,C];
C40B0050-02 [I,A]; C40B0050-06 [I,C]; C40B0050-06 [I,A]

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 6 OF 19 USPATFULL on STN
AN 2007:177114 USPATFULL
TI Genes associate with progression and response in chronic myeloid leukemia and uses thereof
IN Radich, Jerald P., Sammamish, WA, UNITED STATES
Dai, Hongyue, Kenmore, WA, UNITED STATES
Mao, Mao, Kirkland, WA, UNITED STATES
Schelter, Janell M., Bellevue, WA, UNITED STATES
Linsley, Peter S., Seattle, WA, UNITED STATES
PI US 20070154931 A1 20070705
AI US 2006-640517 A1 20061214 (11)
PRAI US 2005-751455P 20051215 (60)
DT Utility
FS APPLICATION
LN.CNT 29037
INCL INCLM: 435/006.000
INCLS: 702/020.000
NCL NCLM: 435/006.000
NCLS: 702/020.000
IC IPCI C12Q0001-68 [I,A]; G06F0019-00 [I,A]
IPCR C12Q0001-68 [I,C]; C12Q0001-68 [I,A]; G06F0019-00 [I,C];
G06F0019-00 [I,A]

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 7 OF 19 USPATFULL on STN
AN 2006:294944 USPATFULL
TI Assays for the detection of biliverdin in birds and reptiles

IN Gregory, Christopher, 1015 Cooper Farm Road, Nicholson, GA, UNITED STATES 30565

Ritchie, Branson W., Athens, GA, UNITED STATES

PI US 20060252110 A1 20061109

AI US 2003-525893 A1 20030827 (10)

WO 2003-US27134 20030827

20050708 PCT 371 date

PRAI US 2002-406175P 20020827 (60)

DT Utility

FS APPLICATION

LN.CNT 1196

INCL INCLM: 435/025.000

NCL NCLM: 435/025.000

IC IPCI C12Q0001-26 [I,A]

IPCR C12Q0001-26 [I,C]; C12Q0001-26 [I,A]

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 8 OF 19 USPATFULL on STN

AN 2005:330597 USPATFULL

TI Identification, monitoring and treatment of disease and characterization of biological condition using gene expression profiles

IN Bevilacqua, Michael P., Boulder, CO, UNITED STATES

Cheronis, John C., Conifer, CO, UNITED STATES

Tryon, Victor, Loveland, CO, UNITED STATES

Bankaitis-Davis, Danute M., Longmont, CO, UNITED STATES

PA Source Precision Medicine, Inc. (U.S. corporation)

PI US 20050287576 A1 20051229

AI US 2005-158504 A1 20050622 (11)

RLI Continuation of Ser. No. US 2002-291856, filed on 8 Nov 2002, PENDING

PRAI US 2001-348213P 20011109 (60)

US 2001-340881P 20011207 (60)

US 2002-369633P 20020403 (60)

US 2002-376997P 20020430 (60)

DT Utility

FS APPLICATION

LN.CNT 4620

INCL INCLM: 435/006.000

INCLS: 702/020.000

NCL NCLM: 435/006.000

NCLS: 702/020.000

IC [7]

ICM C12Q001-68

ICS G06F019-00; G01N033-48; G01N033-50

IPCI C12Q0001-68 [ICM,7]; G06F0019-00 [ICS,7]; G01N0033-48 [ICS,7];

G01N0033-50 [ICS,7]

IPCR G01N0033-68 [I,C*]; G01N0033-68 [I,A]

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 9 OF 19 USPATFULL on STN

AN 2005:286884 USPATFULL

TI Identification, monitoring and treatment of disease and characterization of biological condition using gene expression profiles

IN Bevilacqua, Michael P., Boulder, CO, UNITED STATES

Bankaitis-Davis, Danute M., Longmont, CO, UNITED STATES

Cheronis, John C., Conifer, CO, UNITED STATES

Tryon, Victor, Loveland, CO, UNITED STATES

PA Source Precision Medicine, Inc. (U.S. corporation)

PI US 20050250148 A1 20051110

AI US 2005-159376 A1 20050622 (11)

RLI Continuation of Ser. No. US 2002-291225, filed on 8 Nov 2002, PENDING

Continuation-in-part of Ser. No. US 2001-821850, filed on 29 Mar 2001,

GRANTED, Pat. No. US 6692916 Continuation-in-part of Ser. No. US

2000-605581, filed on 28 Jun 2000, ABANDONED

PRAI US 1999-141542P 19990628 (60)
US 2000-195522P 20000407 (60)
US 2001-348213P 20011109 (60)
US 2001-340881P 20011207 (60)
US 2002-369633P 20020403 (60)
US 2002-376997P 20020430 (60)

DT Utility
FS APPLICATION
LN.CNT 4670

INCL INCLM: 435/006.000
INCLS: 435/091.200

NCL NCLM: 435/006.000
NCLS: 435/091.200

IC [7]
ICM C12Q001-68
ICS C12P019-34
IPCI C12Q0001-68 [ICM,7]; C12P0019-34 [ICS,7]; C12P0019-00 [ICS,7,C*]
IPCR C12P0019-00 [I,C*]; C12P0019-34 [I,A]; C12Q0001-68 [I,C*];
C12Q0001-68 [I,A]; G01N0033-50 [I,C*]; G01N0033-50 [I,A];
G01N0033-574 [I,C*]; G01N0033-574 [I,A]; G01N0033-68 [I,C*];
G01N0033-68 [I,A]

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 10 OF 19 USPATFULL on STN

AN 2004:173188 USPATFULL

TI Identification, monitoring and treatment of disease and characterization
of biological condition using gene expression profiles

IN Bevilacqua, Michael, Boulder, CO, UNITED STATES
Cheronis, John C., Conifer, CO, UNITED STATES
Tryon, Victor, Loveland, CO, UNITED STATES

PI US 20040133352 A1 20040708
US 6960439 B2 20051101

AI US 2002-291225 A1 20021108 (10)

RLI Continuation-in-part of Ser. No. US 2001-821850, filed on 29 Mar 2001,
GRANTED, Pat. No. US 6692916 Continuation-in-part of Ser. No. US
2000-605581, filed on 28 Jun 2000, ABANDONED

PRAI US 2001-348213P 20011109 (60)
US 2001-340881P 20011207 (60)
US 2002-369633P 20020403 (60)
US 2002-376997P 20020430 (60)
US 1999-141542P 19990628 (60)
US 2000-195522P 20000407 (60)

DT Utility
FS APPLICATION
LN.CNT 4839

INCL INCLM: 702/019.000
INCLS: 435/006.000

NCL NCLM: 435/006.000; 702/019.000
NCLS: 702/019.000; 702/020.000

IC [7]
ICM G01N033-48
ICS C12Q001-68
IPCI G01N0033-48 [ICM,7]; C12Q0001-68 [ICS,7]
IPCI-2 C12Q0001-68 [ICM,7]; G06F0019-00 [ICS,7]
IPCR C12P0019-00 [I,C*]; C12P0019-34 [I,A]; C12Q0001-68 [I,C*];
C12Q0001-68 [I,A]; G01N0033-50 [I,C*]; G01N0033-50 [I,A];
G01N0033-574 [I,C*]; G01N0033-574 [I,A]; G01N0033-68 [I,C*];
G01N0033-68 [I,A]

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 11 OF 19 USPATFULL on STN

AN 2004:38576 USPATFULL
 TI Methods of diagnosis of breast cancer, compositions and methods of
 screening for modulators of breast cancer
 IN Mack, David H., Menlo Park, CA, UNITED STATES
 Gish, Kurt C., San Francisco, CA, UNITED STATES
 Afar, Daniel, Brisbane, CA, UNITED STATES
 PA Eos Technology, Inc., South San Francisco, CA, UNITED STATES, 94080-7019
 (U.S. corporation)
 PI US 20040029114 A1 20040212
 AI US 2002-58270 A1 20020124 (10)
 PRAI US 2001-263965P 20010124 (60)
 US 2001-265928P 20010202 (60)
 US 2001-282698P 20010409 (60)
 US 2001-288590P 20010504 (60)
 US 2001-294443P 20010529 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 42494
 INCL INCLM: 435/006.000
 INCLS: 435/069.100; 435/320.100; 435/325.000; 530/350.000; 536/023.500
 NCL NCLM: 435/006.000
 NCLS: 435/069.100; 435/320.100; 435/325.000; 530/350.000; 536/023.500
 IC [7]
 ICM C12Q001-68
 ICS C07H021-04; C07K014-72; C12P021-02; C12N005-06
 IPCI C12Q0001-68 [ICM,7]; C07H0021-04 [ICS,7]; C07H0021-00 [ICS,7,C*];
 C07K0014-72 [ICS,7]; C07K0014-435 [ICS,7,C*]; C12P0021-02
 [ICS,7]; C12N0005-06 [ICS,7]
 IPCR C07K0014-435 [I,C*]; C07K0014-47 [I,A]; C12Q0001-68 [I,C*];
 C12Q0001-68 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 12 OF 19 USPATFULL on STN
 AN 2003:324595 USPATFULL
 TI Methods of diagnosis of Hepatitis C infection, compositions and methods
 of screening for modulators of Hepatitis C infection
 IN Yat Wah Tom, Edward, Sacramento, CA, UNITED STATES
 Zlotnik, Albert, Palo Alto, CA, UNITED STATES
 PA Eos Biotechnology, Inc., South San Francisco, CA (U.S. corporation)
 PI US 20030228570 A1 20031211
 AI US 2003-366435 A1 20030212 (10)
 RLI Continuation of Ser. No. US 2002-206473, filed on 24 Jul 2002, ABANDONED
 PRAI US 2002-366782P 20020321 (60)
 US 2001-308188P 20010726 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 22742
 INCL INCLM: 435/005.000
 INCLS: 435/006.000; 435/069.300; 435/320.100; 435/325.000; 530/350.000;
 530/388.300; 536/023.720
 NCL NCLM: 435/005.000
 NCLS: 435/006.000; 435/069.300; 435/320.100; 435/325.000; 530/350.000;
 530/388.300; 536/023.720
 IC [7]
 ICM C12Q001-70
 ICS C12Q001-68; C07H021-04; C07K014-02; C07K016-08; C12P021-02;
 C12N005-06
 IPCI C12Q0001-70 [ICM,7]; C12Q0001-68 [ICS,7]; C07H0021-04 [ICS,7];
 C07H0021-00 [ICS,7,C*]; C07K0014-02 [ICS,7]; C07K0014-005
 [ICS,7,C*]; C07K0016-08 [ICS,7]; C12P0021-02 [ICS,7]; C12N0005-06
 [ICS,7]
 IPCR C12Q0001-70 [I,C*]; C12Q0001-70 [I,A]

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 13 OF 19 USPATFULL on STN
AN 2003:312174 USPATFULL
TI Identification, monitoring and treatment of disease and characterization
of biological condition using gene expression profiles
IN Bevilacqua, Michael, Boulder, CO, UNITED STATES
Cheronis, John C., Conifer, CO, UNITED STATES
Tryon, Victor, Loveland, CO, UNITED STATES
PI US 20030219771 A1 20031127
US 6964850 B2 20051115
AI US 2002-291856 A1 20021108 (10)
PRAI US 2001-348213P 20011109 (60)
US 2001-340881P 20011207 (60)
US 2002-369633P 20020403 (60)
US 2002-376997P 20020430 (60)
DT Utility
FS APPLICATION
LN.CNT 4844
INCL INCLM: 435/006.000
INCLS: 702/020.000
NCL NCLM: 435/006.000
NCLS: 702/019.000; 702/020.000
IC [7]
ICM C12Q001-68
ICS G06F019-00; G01N033-48; G01N033-50
IPCI C12Q0001-68 [ICM,7]; G06F0019-00 [ICS,7]; G01N0033-48 [ICS,7];
G01N0033-50 [ICS,7]
IPCI-2 C12Q0001-68 [ICM,7]; G06F0019-00 [ICS,7]
IPCR G01N0033-68 [I,C*]; G01N0033-68 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 14 OF 19 USPATFULL on STN
AN 2003:220740 USPATFULL
TI Methods and compositions for diagnosing and treating rheumatoid
arthritis
IN Pittman, Debra D., Windham, NH, UNITED STATES
Feldman, Jeffrey L., Arlington, MA, UNITED STATES
Shields, Kathleen M., Harvard, MA, UNITED STATES
Trepicchio, William L., Andover, MA, UNITED STATES
PI US 20030154032 A1 20030814
AI US 2001-23451 A1 20011217 (10)
PRAI US 2000-255861P 20001215 (60)
DT Utility
FS APPLICATION
LN.CNT 25385
INCL INCLM: 702/020.000
NCL NCLM: 702/020.000
IC [7]
ICM G06F019-00
ICS G01N033-48
IPCI G06F0019-00 [ICM,7]; G01N0033-48 [ICS,7]
IPCR A61K0038-00 [N,C*]; A61K0038-00 [N,A]; C07K0014-435 [I,C*];
C07K0014-47 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 15 OF 19 USPATFULL on STN
AN 2002:301655 USPATFULL
TI Compounds and methods for regulating cell differentiation
IN Falchuk, Kenneth H., Newton, MA, UNITED STATES
PA President & Fellows of Harvard College, Cambridge, MA, UNITED STATES
(U.S. corporation)

PI US 20020169201 A1 20021114
 US 6902881 B2 20050607
 AI US 2001-8356 A1 20011113 (10)
 RLI Continuation-in-part of Ser. No. US 2001-977866, filed on 15 Oct 2001,
 PENDING
 PRAI US 2000-240497P 20001013 (60)
 US 2000-247299P 20001110 (60)
 US 2001-262233P 20010117 (60)
 US 2001-264814P 20010129 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 4893
 INCL INCLM: 514/422.000
 INCLS: 548/518.000
 NCL NCLM: 435/001.100; 514/422.000
 NCLS: 435/325.000; 514/359.000; 514/422.000; 548/518.000
 IC [7]
 ICM A61K031-4025
 ICS C07D043-14
 IPCI A61K0031-4025 [ICM,7]; C07D0043-14 [ICS,7]
 IPCI-2 A01N0001-00 [ICM,7]; A01N0043-38 [ICS,7]; A01N0043-34 [ICS,7,C*];
 C12N0005-02 [ICS,7]; A61K0031-409 [ICS,7]
 IPCR A61K0031-409 [I,C*]; A61K0031-409 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 16 OF 19 USPAT2 on STN
 AN 2007:17432 USPAT2
 TI Primary rat hepatocyte toxicity modeling
 IN Mendrick, Donna L., Gaithersburg, MD, UNITED STATES
 Porter, Mark W., Gaithersburg, MD, UNITED STATES
 Johnson, Kory R., Gaithersburg, MD, UNITED STATES
 Higgs, Brandon, Gaithersburg, MD, UNITED STATES
 Castle, Arthur L., Gaithersburg, MD, UNITED STATES
 Orr, Michael, Gaithersburg, MD, UNITED STATES
 Elashoff, Michael R., Gaithersburg, MD, UNITED STATES
 PA Ocimum Biosolutions, Inc., Indianapolis, IN, UNITED STATES (U.S.
 corporation)
 PI US 7469185 B2 20081223
 AI US 2003-357507 20030204 (10)
 PRAI US 2002-407688P 20020904 (60)
 US 2002-394230P 20020709 (60)
 US 2002-394253P 20020709 (60)
 US 2002-378653P 20020509 (60)
 US 2002-378665P 20020509 (60)
 US 2002-378652P 20020509 (60)
 US 2002-378370P 20020508 (60)
 US 2002-374139P 20020422 (60)
 US 2002-373602P 20020419 (60)
 US 2002-373601P 20020419 (60)
 US 2002-371413P 20020411 (60)
 US 2002-371150P 20020410 (60)
 US 2002-371135P 20020410 (60)
 US 2002-371134P 20020410 (60)
 US 2002-370248P 20020408 (60)
 US 2002-363534P 20020313 (60)
 US 2002-353171P 20020204 (60)
 DT Utility
 FS GRANTED
 LN.CNT 44495
 INCL INCLM: 702/019.000
 INCLS: 435/006.000; 700/030.000; 702/022.000; 707/104.100
 NCL NCLM: 702/019.000; 435/006.000

NCLS: 435/006.000; 700/030.000; 702/022.000; 707/999.107; 702/020.000
IC IPCI C12Q0001-68 [I,A]; G06F0019-00 [I,A]; G01N0033-48 [I,A];
G01N0033-50 [I,A]
IPCI-2 G06F0019-00 [I,A]
IPCR G06F0019-00 [I,C]; G06F0019-00 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 17 OF 19 USPAT2 on STN
AN 2004:173188 USPAT2
TI Identification, monitoring and treatment of disease and characterization
of biological condition using gene expression profiles
IN Bevilacqua, Michael, Boulder, CO, UNITED STATES
Bankaitis-Davis, Danute M., Longmont, CO, UNITED STATES
Cheronis, John C., Conifer, CO, UNITED STATES
Tryon, Victor, Loveland, CO, UNITED STATES
PA Source Precision Medicine, Inc., Boulder, CO, UNITED STATES (U.S.
corporation)
PI US 6960439 B2 20051101
AI US 2002-291225 20021108 (10)
RLI Continuation-in-part of Ser. No. US 2001-821850, filed on 29 Mar 2001,
Pat. No. US 6692916 Continuation-in-part of Ser. No. US 2000-605581,
filed on 28 Jun 2000, ABANDONED
PRAI US 2001-348213P 20011109 (60)
US 2001-340881P 20011207 (60)
US 2002-369633P 20020403 (60)
US 2002-376997P 20020430 (60)
US 1999-141542P 19990628 (60)
US 2000-195522P 20000407 (60)
DT Utility
FS GRANTED
LN.CNT 4579
INCL INCLM: 435/006.000
INCLS: 702/019.000; 702/020.000
NCL NCLM: 435/006.000; 702/019.000
NCLS: 702/019.000; 702/020.000
IC [7]
ICM C12Q001-68
ICS G06F019-00
IPCI G01N0033-48 [ICM,7]; C12Q0001-68 [ICS,7]
IPCI-2 C12Q0001-68 [ICM,7]; G06F0019-00 [ICS,7]
IPCR C12P0019-00 [I,C*]; C12P0019-34 [I,A]; C12Q0001-68 [I,C*];
C12Q0001-68 [I,A]; G01N0033-50 [I,C*]; G01N0033-50 [I,A];
G01N0033-574 [I,C*]; G01N0033-574 [I,A]; G01N0033-68 [I,C*];
G01N0033-68 [I,A]
EXF 435/6; 702/19; 702/20
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 18 OF 19 USPAT2 on STN
AN 2003:312174 USPAT2
TI Identification, monitoring and treatment of disease and characterization
of biological condition using gene expression profiles
IN Bevilacqua, Michael P., Boulder, CO, UNITED STATES
Cheronis, John C., Conifer, CO, UNITED STATES
Tryon, Victor, Loveland, CO, UNITED STATES
Bankaitis-Davis, Danute M., Longmont, CO, UNITED STATES
PA Source Precision Medicine, Inc., Boulder, CO, UNITED STATES (U.S.
corporation)
PI US 6964850 B2 20051115
AI US 2002-291856 20021108 (10)
PRAI US 2002-376997P 20020430 (60)
US 2002-369633P 20020403 (60)
US 2001-340881P 20011207 (60)

US 2001-348213P 20011109 (60)
 DT Utility
 FS GRANTED
 LN.CNT 4683
 INCL INCLM: 435/006.000
 INCLS: 702/019.000; 702/020.000
 NCL NCLM: 435/006.000
 NCLS: 702/019.000; 702/020.000
 IC [7]
 ICM C12Q001-68
 ICS G06F019-00
 IPCI C12Q0001-68 [ICM,7]; G06F0019-00 [ICS,7]; G01N0033-48 [ICS,7];
 G01N0033-50 [ICS,7]
 IPCI-2 C12Q0001-68 [ICM,7]; G06F0019-00 [ICS,7]
 IPCR G01N0033-68 [I,C*]; G01N0033-68 [I,A]
 EXF 702/19; 702/20; 435/6
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 19 OF 19 USPAT2 on STN
 AN 2002:301655 USPAT2
 TI Compounds and methods for regulating cell differentiation
 IN Falchuk, Kenneth H., Newton, MA, UNITED STATES
 PA President and Fellows of Harvard College, Cambridge, MA, UNITED STATES
 (U.S. corporation)
 PI US 6902881 B2 20050607
 AI US 2001-8356 20011113 (10)
 RLI Continuation-in-part of Ser. No. US 2001-977866, filed on 15 Oct 2001,
 PENDING
 PRAI US 2001-264814P 20010129 (60)
 US 2001-262233P 20010117 (60)
 US 2000-247299P 20001110 (60)
 US 2000-240497P 20001013 (60)
 DT Utility
 FS GRANTED
 LN.CNT 4994
 INCL INCLM: 435/001.100
 INCLS: 435/325.000; 514/359.000; 514/422.000
 NCL NCLM: 435/001.100; 514/422.000
 NCLS: 435/325.000; 514/359.000; 514/422.000; 548/518.000
 IC [7]
 ICM A01N001-00
 ICS A01N043-38; C12N005-02; A61K031-409
 IPCI A61K0031-4025 [ICM,7]; C07D0043-14 [ICS,7]
 IPCI-2 A01N0001-00 [ICM,7]; A01N0043-38 [ICS,7]; A01N0043-34 [ICS,7,C*];
 C12N0005-02 [ICS,7]; A61K0031-409 [ICS,7]
 IPCR A61K0031-409 [I,C*]; A61K0031-409 [I,A]
 EXF 435/1.1; 435/325; 514/359; 514/422
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> s L6 and detect?(p)biliverdin
 L7 4 L6 AND DETECT?(P) BILIVERDIN

=> d 17 1-4

L7 ANSWER 1 OF 4 IFIPAT COPYRIGHT 2010 IFI on STN
 AN 11880348 IFIPAT;IFIUDB;IFICDB
 TI Advanced drug development and manufacturing; Using x-ray fluorescence to
 monitor protein ligand binding; rational drug design and screening
 IN Baldwin Sharon M; Berger Jennifer A; Birnbaum Eva R; Burrell Anthony K;
 Harris Michael N; Koppisch Andrew T; McCleskey T Mark; Stewart Jeffrey
 Joseph; Warner Benjamin P

PA Unassigned Or Assigned To Individual (68000)
 PPA LOS ALAMOS NATIONAL SECURITY LLC (Probable)
 PI US 20080220441 A1 20080911
 AI US 2007-974156 20071010 (11)
 RLI US 2001-859701 20010516 CONTINUATION-IN-PART PENDING
 US 2002-206524 20020725 CONTINUATION-IN-PART ABANDONED
 US 2003-621825 20030716 CONTINUATION-IN-PART 6858148
 PRAI US 2006-850594P 20061010 (Provisional)
 FI US 20080220441 20080911
 US 6858148
 DT Utility; Patent Application - First Publication
 FS CHEMICAL
 APPLICATION
 OS CA 149:326754
 ED Entered STN: 17 Sep 2008
 Last Updated on STN: 16 Mar 2009
 CLMN 48

L7 ANSWER 2 OF 4 USPATFULL on STN
 AN 2006:294944 USPATFULL
 TI Assays for the detection of biliverdin in birds and
 reptiles
 IN Gregory, Christopher, 1015 Cooper Farm Road, Nicholson, GA, UNITED
 STATES 30565
 Ritchie, Branson W., Athens, GA, UNITED STATES
 PI US 20060252110 A1 20061109
 AI US 2003-525893 A1 20030827 (10)
 WO 2003-US27134 20030827
 20050708 PCT 371 date
 PRAI US 2002-406175P 20020827 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 1196
 INCL INCLM: 435/025.000
 NCL NCLM: 435/025.000
 IC IPCI C12Q0001-26 [I,A]
 IPCR C12Q0001-26 [I,C]; C12Q0001-26 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 3 OF 4 USPATFULL on STN
 AN 2002:301655 USPATFULL
 TI Compounds and methods for regulating cell differentiation
 IN Falchuk, Kenneth H., Newton, MA, UNITED STATES
 PA President & Fellows of Harvard College, Cambridge, MA, UNITED STATES
 (U.S. corporation)
 PI US 20020169201 A1 20021114
 US 6902881 B2 20050607
 AI US 2001-8356 A1 20011113 (10)
 RLI Continuation-in-part of Ser. No. US 2001-977866, filed on 15 Oct 2001,
 PENDING
 PRAI US 2000-240497P 20001013 (60)
 US 2000-247299P 20001110 (60)
 US 2001-262233P 20010117 (60)
 US 2001-264814P 20010129 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 4893
 INCL INCLM: 514/422.000
 INCLS: 548/518.000
 NCL NCLM: 435/001.100; 514/422.000
 NCLS: 435/325.000; 514/359.000; 514/422.000; 548/518.000
 IC [7]

ICM A61K031-4025
ICS C07D043-14
IPCI A61K0031-4025 [ICM,7]; C07D0043-14 [ICS,7]
IPCI-2 A01N0001-00 [ICM,7]; A01N0043-38 [ICS,7]; A01N0043-34 [ICS,7,C*];
C12N0005-02 [ICS,7]; A61K0031-409 [ICS,7]
IPCR A61K0031-409 [I,C*]; A61K0031-409 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 4 OF 4 USPAT2 on STN
AN 2002:301655 USPAT2
TI Compounds and methods for regulating cell differentiation
IN Falchuk, Kenneth H., Newton, MA, UNITED STATES
PA President and Fellows of Harvard College, Cambridge, MA, UNITED STATES
(U.S. corporation)
PI US 6902881 B2 20050607
AI US 2001-8356 20011113 (10)
RLI Continuation-in-part of Ser. No. US 2001-977866, filed on 15 Oct 2001,
PENDING
PRAI US 2001-264814P 20010129 (60)
US 2001-262233P 20010117 (60)
US 2000-247299P 20001110 (60)
US 2000-240497P 20001013 (60)
DT Utility
FS GRANTED
LN.CNT 4994
INCL INCLM: 435/001.100
INCLS: 435/325.000; 514/359.000; 514/422.000
NCL NCLM: 435/001.100; 514/422.000
NCLS: 435/325.000; 514/359.000; 514/422.000; 548/518.000
IC [7]
ICM A01N001-00
ICS A01N043-38; C12N005-02; A61K031-409
IPCI A61K0031-4025 [ICM,7]; C07D0043-14 [ICS,7]
IPCI-2 A01N0001-00 [ICM,7]; A01N0043-38 [ICS,7]; A01N0043-34 [ICS,7,C*];
C12N0005-02 [ICS,7]; A61K0031-409 [ICS,7]
IPCR A61K0031-409 [I,C*]; A61K0031-409 [I,A]
EXF 435/1.1; 435/325; 514/359; 514/422
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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L7 ANSWER 1 OF 4 IFIPAT COPYRIGHT 2010 IFI on STN
ACLM . . . to measurement, the solution comprising a buffer, the buffer
being substantially free of at least one of the chemicals or
functional groups selected from the group of dimethylsulfoxide,
thiols, sulfate anion, sulfonate anions, chloride anion, bromide anion,
fluoride anion, iodide anion,. . .
. . . solution prior to measurement, the solution comprising a buffer, and
the buffer comprises one or more of the chemicals or functional
groups selected from the group of amine, imine, nitrate anion, nitrite
anion, ammonium cation, and iminium cation.
. . . claim 1, wherein the receptor comprises at least one of the receptors
selected from the list of 1,3,4,6-Tetrachloro-1,4-Cyclohexadiene H;
1,3,6,8-Tetrahydroxynaphthalene Reductase;
1,3-1,4-Beta-Glucanase; 1,4-Alpha Maltotetrahydrolase; 1,4-Alpha-D-Glucan
Glucanohydrolase; 1,4-Beta-D-Glucan Cellobiohydrolase Cel7;
1,4-Beta-D-Glucan Cellobiohydrolase I; 1,4-dihydropyridine Receptor on
alpha1 subunit of L-type voltage sensitive Ca2+ channels; 10 Kda
Chaperonin; 10-Formyltetrahydrofolate Dehydrogenase; 11-cis retinol
dehydrogenase; 12-Oxophytodienoate Reductase;
12-Oxophytodienoate Reductase 1; 12-Oxophytodienoate-10,11-

Reductase; 14-3-3-Like Protein C; 15-hydroxyprostaglandin dehydrogenase (NAD+); 17-Beta-Hydroxysteroid Dehydrogenase; 17-Beta-Hydroxysteroid Dehydrogenase 4; 17 Kd Fetal Brain Protein; 19-Mer Peptide Fragment Of. . . OKT3 Heavy Chain 1; 1SY6:L OKT3 Light Chain 1; 2,2-Dialkylglycine Decarboxylase; 2,3-Bisphosphoglycerate-Independent Phosphog; 2, 3-Dihydroxybenzoate-Amp Ligase; 2,3-Dihydroxybiphenyl Dioxygenase; 2,3-Dihydroxybiphenyl-1,2-Dioxygenase; 2,4-Dienoyl-Coa Reductase; 2,5-Diketo-D-Gluconic Acid Reductase; 23-Kda Polypeptide Of Photosystem II Oxygen-; 23S Ribosomal; 23S Ribosomal RNA; 23S rRNA of 50S ribosomal subunit; 25-hydroxyvitamin D-1 alpha. . . Type 3; 3-Alpha-Hydroxysteroid/Dihydrodiol Dehydrogease; 3-Carboxy-Cis,Cis-Muconate Cycloisomerase; 3-Dehydroquinone Dehydratase; 3-Dehydroquinone Dehydratase Arod; 3-Dehydroquinone Synthase; 3-Deoxy-D-Arabetio-Heptulosonate-7-Phosphatase; 3-Deoxy-Manno-Octulosonate Cytidyltransfer; 3-Hydroxy-3-Methylglutaryl-Coa Synthase; 3-Hydroxy-3-Methylglutaryl-Coenzyme A Reductase; 3-Hydroxyacyl-Coa Dehydrogenase; 3-Hydroxyacyl-Coa Dehydrogenase Type 11; 3-hydroxyisobutyrate dehydrogenase, mitochondrial precursor; 3-Isopropylmalate Dehydrogenase; 3-Ketoacetyl-Coa Thiolase; 3-Keto-L-Gulonate 6-Phosphate Decarboxylase; 3-keto-steroid reductase; 3-Mercaptopyruvate Sulfurtransferase; 3-Methyl-2-Oxobutanoate Hydroxymethyltra; 3-Methyl-2-Oxobutanoate Hydroxymethyltransfe; 3-Methyladenine DNA Glycosylase; 3-Methylaspartate Ammonia-Lyase; 3-oxo-5-alpha-steroid 4-dehydrogenase 2; 3-Oxoacyl-; 3-Oxoacyl-(Acyl-Carrier Protein) Reductase; 3-Oxoacyl-(Acyl-Carrier Protein) Synthas; 3-Oxoacyl-(Acyl-Carrier-Protein) Synthas; 3-oxoacyl-(acyl-carrier-protein) synthase I; 3-oxoacyl-(acyl-carrier-protein) synthase II; 3-oxoacyl-(acyl-carrier-protein) synthase III; 3-Phosphoglycerate Kinase; 3-Phosphoinositide Dependent Protein Kin; . . . 4-Hydroxythreonine-4-Phosphate Dehydrogenase; 4M5. 3 Anti-Fluorescein Single Chain Antibody; 4-Oxalocrotonate Tautomerase; 4'-Phosphopantetheinyl Transferase Sfp; 4-trimethylaminobutyraldehyde dehydrogenase; 5,10-Methenyltetrahydrofolate Synthetase; 5,10-Methylenetetrahydrofolate Dehydrogenase; 5,10-Methylenetetrahydrofolate Reductase; 50S Ribosomal Protein L1P; 50S subunit of the 70S ribosome of bacteria; 5-alpha reductase 1; 5-Aminolaevulinic Acid Dehydratase; 5-aminolevulinate synthase; 5-Aminolevulinic Acid Dehydratase; 5'-AMP-activated protein kinase, beta-2 subunit; 5'-AMP-activated protein kinase, catalytic alpha-1 chain; . . . Tetrahydropterin Synthase; 7 Alpha-Hydroxysteroid Dehydrogenase; 7,8-Diamino-Pelargonic Acid Aminotransferase; 7, 8-Dihydro-6-Hydroxymethylpterin-Pyrophosp; 70 Kilodalton Heat Shock Protein; 70-Kda Soluble Lytic Transglycosylase; 7-dehydrocholesterol reductase; 8-Amino-7-Oxononanoate Synthase; 8-Oxoguanine DNA Glycosylase; 92 Kda Type IV Collagenase; A chain; A/G-Specific Adenine Glycosylase; Aac; Aah2: Lqh-Alpha-It; Abc Transporter, ATP Binding Protein; Acetate Kinase; Acetoacetyl-Coa Thiolase; Acetohydroxy-Acid Isomeroeductase; Acetohydroxy-Acid Synthase; Acetoin Reductase; Acetolactate Synthase, Catabolic; Acetolactate Synthase, Mitochondrial; Acetyl Group; Acetyl Transferase; Acetyl Xylan Esterase; Acetylcholine Binding Protein; Acetylcholine-Binding Protein; Acetylcholinesterase; Acetylcholinesterase;. . . Protein Heart Isoform T1; ADP,ATP carrier protein, fibroblast isoform; ADP,ATP carrier protein, heart/skeletal muscle isoform T1; ADP, ATP carrier protein, liver isoform T2; ADP-Dependent Glucokinase; ADP-L-Glycero-D-Mannoheptose 6-Epimerase; Adpr Pyrophosphatase; ADP-Ribose Pyrophosphatase; ADP-Ribosyl Cyclase; ADP-Ribosylation Factor 1; ADP-Ribosylation Factor 2; ADP-Ribosylation Factor. . . Factor 6; ADP-Ribosylation Factor-Like 8; ADP-Ribosylation Factor-Like Protein 1; ADP-Ribosylation Factor-Like Protein 3;

ADP-Ribosylation Factor-Like Protein 5; ADP-Ribosyltransferase;
 Adrenodoxin; Adrenodoxin Reductase; Adsorption Protein P2;
 . . . dimeric NADP-preferring; Aldehyde Dehydrogenase, Mitochondrial
 Precur; Aldehyde dehydrogenase, mitochondrial precursor; Aldehyde
 Ferredoxin Oxidoreductase Protein C; Aldehyde oxidase; Aldehyde
 Oxidoreductase; Aldehyde Reductase; Aldo-Keto Reductase
 Family 1 Member C1; Aldo-keto reductase family 1 member C2;
 Aldo-Keto Reductase Family 1 Member C3; Aldo-keto
 reductase family 1 member C4; Aldolase; Aldolase Protein; Aldose
 1-Epimerase; Aldose Reductase; Alginate Lyase; Algq1; Algq2;
 ALK tyrosine kinase receptor; Alkaline Phosphatase; Alkaline Phosphatase;
 Alkyl Hydroperoxide Reductase Subunit F; Allantoate
 Amidohydrolase; Allene Oxide Synthase-Lipoxygenase Protein; Alliin Lyase;
 Alpha Amylase; Alpha Glutathione S-Transferase; Alpha,
 Alpha-Trehalose-Phosphate Synthase; Alpha-1 Catenin;. . . synthase
 (Fragment); Argininosuccinate Synthetase; Arginosuccinate lyase;
 Arginosuccinate synthase; Aristolochene Synthase; Arnb Aminotransferase;
 Arno; Aromatase; Aromatic Amino Acid Aminotransferase; Arpg836; Arsenate
 Reductase; Arsenical pump-driving ATPase; Arsenical Resistance
 Operon Repressor, Pu; Arsenite-Translocating Atpase; Arthropodan
 Hemocyanin; Artificial Nucleotide Binding Protein; Artocarpin; Aryl
 Sulfotransferase; Arylamine. . . 11; Atrial Natriuretic Peptide
 Clearance Recepto; Atrial natriuretic peptide receptor A; Atrial
 Natriuretic Peptide Receptor A; Atrolysin C; Augmenter Of Liver
 Regeneration; Aurora-Related Kinase 1; Autocrine Motility Factor;
 Autoinducer-2 Production Protein Luxs; Autolysin; Auxin Binding Protein
 1; Avermectin-Sensitive Chloride Channel GI; Avian Sarcoma
 Virus Integrase; Avidin; Axin; Azurin; B chain; B Lymphocyte Stimulator;
 B4-Dimer; B9340; Bacterial azoreductase (Bacillus sp); Bacterial
 isoleucyl-tRNA synthetase;. . . Protein 4; Bap1; Basement Membrane
 Protein Bm-40; Basic Fibroblast Growth Factor; Basic Phospholipase A2;
 Bbal; Bba5; B-cell receptor; Benzoate 1,2-Dioxygenase Reductase
 ; Benzodiazepine Receptor; Benzoylformate Decarboxylase; Benzyl Alcohol
 Dehydrogenase; Beta 1 adrenergic receptor; Beta 1,4
 Galactosyltransferase; Beta 2 adrenergic receptor; beta chain. . .
 Beta-Hordothionin; Beta-Hydroxydecanoyl Thiol Ester Dehydrase;
 Betaine-Homocysteine Methyltransferase; Betaine-Homocysteine
 S-Methyltransferase; Beta-Keto Acyl Carrier Protein Reductase;
 Beta-Ketoacyl (Acyl Carrier Protein) Synthas;
 Beta-Ketoacyl-(Acyl-Carrier-Protein) Synthas; Beta-Ketoacyl-Acp Synthase
 III; Beta-Ketoacyl-Acyl Carrier Protein Synth; Beta-Ketoacyl-Acyl Carrier
 Protein Synthase; Beta-Ketoacylsynthase III;. . . Beta-Spectrin;
 Beta-Trypsin; Beta-Tryptase; Bh0236 Protein; Bifunctional
 3'-Phosphoadenosine 5'-Phospho; Bifunctional adenosylcobalamin
 biosynthesis protein cobU; Bifunctional aminoacyl-tRNA synthetase;
 Bifunctional Deaminase/Diphosphatase; Bifunctional Dihydrofolate
 Reductase-Thymidy; Bifunctional dihydrofolate reductase
 -thymidylate synthase; Bifunctional Histidine Biosynthesis Prot;
 Bifunctional methylenetetrahydrofolate dehydrogenase/ cyclohydrolase;
 Bifunctional P450: Nadph-P450 Reductase; Bifunctional PGK/TIM
 (Includes: Phosphoglycerate kinase, EC 2.7. 2.3, Triosephosphate
 isomerase, EC 5.3.1.1, TIM, Triose-phosphate isomerase); Bifunctional
 Purine Biosynthesis Protein Pur;. . . Rela/Spot; Bikunin; Bile Acid
 Receptor; Bile salt export pump; Bile-Salt Activated Lipase; Biliary
 Glycoprotein C; Bilin Binding Protein; Bilin-Binding Protein;
 Biliverdin Ix Beta Reductase; Biliverdin
 Reductase A; Biliverdin reductase A
 precursor; Bioh Protein; Biosynthetic Thiolase; Biotin Synthase;
 biotinidase; biotin-protein ligase; Biphenyl-2,3-Diol 1,2-Dioxygenase;
 Bleomycin Resistance Determinant; Bleomycin Resistance Protein;
 Bleomycin-Binding. . . Anhydrase; Carbonic Anhydrase I; Carbonic
 Anhydrase II; Carbonic Anhydrase III; Carbonic Anhydrase IV; Carbonic

Anhydrase Xii; Carbonic Anhydrase Xiv; Carbonyl Reductase;
 Carbonyl Reductase (Nadph) 1; Carboxy Methyl Transferase For
 Protein Phosp; Carboxy-Cis,Cis-Muconate Cyclase; Carboxyethylarginine
 Synthase; Carboxylesterase; Carboxylesterase Est2; Carboxylesterase
 Precursor; Carboxymethylated Rhodanese; Carboxymuconolactone. . . M;
 Carboxy-Terminal Domain RNA Polymerase I; Caminomycin
 4-O-Methyltransferase; Carnitine Acetyltransferase; Carnitine
 Acetyltransferase Isoform 2; Carnitine O-acetyltransferase; Carnitine
 O-palmitoyltransferase I, mitochondrial liver isoform (CPT-1);
 Carnitine O-palmitoyltransferase II, mitochondrial (CPT-2); Casein Kinase
 II, Alpha Chain; Casein Kinase-1; Caspase-1 precursor; Caspase-3;
 Caspase-7; Catabolic Alanine. . . Binding Protein Ab80; Chlorophyll
 A-B Binding Protein, Chloroplast; Chloroplast Ferredoxin-Nadp+
 Oxidoreductase; Chloroplast Outer Envelope Protein Oep34; Chloroplastic
 Ascorbate Peroxidase; Cho Reductase; Cholecystokinin type A
 receptor; Cholera Toxin; Cholera Toxin B Subunit; Cholesterol Esterase;
 Cholesterol Oxidase; Choline dehydrogenase; Choline kinase alpha;
 Choline. . .
 . . . Conserved Protein Mth1675; Constitutive Androstane Receptor;
 Contryphan-R; Contryphan-Sm; Contryphan-Vn, Major Form; Copper Amine
 Oxidase; Copper Amine Oxidase; Copper Amine Oxidase, Liver
 Isozyme; Copper Transport Protein Atox1; Copper-Containing Nitrite
 Reductase; Core Protein; Corticosteroid 11-Beta-Dehydrogenase
 Isozyme; Corticosteroid 11-Beta-Dehydrogenase, Isozym; Corticosteroid
 11-beta-dehydrogenase, isozyme 1; Corticosteroid 11-beta-dehydrogenase,
 isozyme 2; Corticosteroid Receptor; Corticotropin Releasing. . .
 Cystic Fibrosis Transmembrane Conductanc; Cystic Fibrosis Transmembrane
 Conductance Re; Cystine/glutamate transporter; Cystinosin; Cytidine
 Deaminase; Cytidine Monophospho-N-Acetylneuraminic Acid; Cytidylate
 Kinase; Cytochrome B=5=Reductase; Cytochrome B2; Cytochrome B2,
 Mitochondrial; Cytochrome B5; Cytochrome B5 Outer Mitochondrial Membrane
 Is; Cytochrome B562; Cytochrome C; Cytochrome C'; Cytochrome C'';
 Cytochrome C Family Protein; Cytochrome C Nitrite Reductase;
 Cytochrome c oxidase subunit 1; Cytochrome C Peroxidase; Cytochrome C
 Peroxidase, Mitochondrial; Cytochrome C, Iso-1; Cytochrome C, Putative;
 Cytochrome C2;. . . Cytochrome C551 Peroxidase; Cytochrome C552;
 Cytochrome C-552; Cytochrome C-553; Cytochrome C-554; Cytochrome C-556;
 Cytochrome C6; Cytochrome C7; Cytochrome Cdl Nitrite Reductase;
 Cytochrome CI; Cytochrome F; Cytochrome Oxidase Subunit II; Cytochrome
 P450; Cytochrome P450 107A1; Cytochrome P450 119; Cytochrome P450 121;
 Cytochrome. . . Residue Peptide; De Novo Designed Cyclic Peptide;
 Deacetoxycephalosporin C Synthase; Death-Associated Protein Kinase;
 Death-Associated Protein Kinase 1; Decorin; Dehaloperoxidase;
 Dehydrogenase/ reductase SDR family member 4; Delta 2
 Crystallin; Delta Crystallin I; Delta Crystallin II;
 Delta-1-pyrroline-5-carboxylate dehydrogenase, mitochondrial precursor;
 Delta-Aminolevulinic Acid Dehydratase;. . . Diaminopimelate
 Decarboxylase; Dianthin 30; Dienelactone Hydrolase; Dienoyl-CoA
 Isomerase; Digal16; Di-Haem Cytochrome C Peroxidase; Diheme Cytochrome C
 Napb; Di-Heme Peroxidase; Dihydrodipicolinate Reductase;
 Dihydrodipicolinate Reductase; Dihydrodipicolinate Synthase;
 Dihydrofolate Reductase; Dihydrofolate Reductase
 (malarial); Dihydrolipoamide Dehydrogenase; Dihydrolipoyl dehydrogenase,
 mitochondrial precursor; Dihydrolipoyl Transacetylase;
 Dihydrolipoyllsine-residue acetyltransferase component of pyruvate
 dehydrogenase complex, mitochondrial precursor; Dihydroneopterin
 Aldolase; Dihydroorotate; Dihydroorotate Dehydrogenase; Dihydroorotate
 Dehydrogenase A; Dihydroorotate Dehydrogenase, mitochondrial (Precursor);
 dihydropterate synthase (bacterial); Dihydropteridine Reductase
 ; Dihydropteridine Reductase; Dihydropteroate synthase;
 Dihydropteroate Synthase (malarial); Dihydropteroate synthase
 (Pneumocystis carinii); Dihydropteroate Synthase I; Dihydropyridine

calcium channel; Dihydropyridine-sensitive L-type, calcium channel
alpha-2/delta subunits; Dihydropyrimidine dehydrogenase; Dihydroxyacetone
Kinase; Diisopropylfluorophosphatase; Dimeric Hemoglobin; Dimethyl
Sulfoxide Reductase; Dipeptidyl Aminopeptidase-Like Protein 6;
Dipeptidyl Peptidase I Dipeptidyl Peptidase IV; Dipeptidyl Peptidase IV;
Dipeptidyl Peptidase IV Soluble Form; Diphtheria Toxin;. . .
Diphtheria Toxin Repressor; Diphthine Synthase; Dissimilatory
Copper-Containing Nitrite; Dissimilatory Copper-Containing Nitrite Redu;
D-lactate dehydrogenase; D-Lactate Dehydrogenase; Dlp-1;
D-Maltodextrin-Binding Protein; DmsO Reductase; DNA Adenine
Methylase; DNA Beta-Glucosyltransferase; DNA Cytosine Methyltransferase
Dnmt2; DNA Double-Strand Break Repair Rad50 Atpase; DNA Gyrase B; DNA
gyrase. . .
. . . Complexed With Beta; D-Ribose-Binding Protein Mutant With Gly 134;
D-Ribose-Binding Protein Mutant With Ile 132; Drosophila Neuroglian;
Dtdp-4-Dehydrorhamnose 3,5-Epimerase; Dtdp-4-Dehydrorhamnose
Reductase, Rfbd O; Dtdp-6-Deoxy-D-Xylo-4-Hexylose 3,5-Epimerase;
Dtdp-D-Glucose 4,6-Dehydratase; Dtdp-Glucose Oxidoreductase; Dual Adaptor
Of Phosphotyrosine and 3-Phosp; Dual Specificity Mitogen-Activated
Protein K; Dual Specificity Protein Kinase Clk1; Duck Ovotransferrin;
Duodenase; Dutp Pyrophosphatase; D-Xylose Isomerase; E. coli citrate
synthetase; E. coli glutathione reductase; E. coli malate
dehydrogenase; E. Coli Maltodextrin Phosphorylase; E. coli pyruvate
dehydrogenase; E. coli ribosomal proteins; Eafp 2; Early Endosomal. . .
receptor precursor; Endothiapepsin; Endothiapepsin precursor;
Endoxylanase; Endoxylanase 11A; Engrailed Homeodomain; Enhancing Lycopene
Biosynthesis Protein; Enkephalinase; Enolase; Enolase 1; Enoyl Acp
Reductase; Enoyl Acyl Carrier Protein Reductase;
Enoyl-(Acyl-Carrier Protein) Reductase;
Enoyl-(Acyl-Carrier-Protein) Reductase;
Enoyl-(Acyl-Carrier-Protein) Reductase;
Enoyl-(Acyl-Carrier-Protein) Reductase (Nadh;
Enoyl-(acyl-carrier-protein) reductase (NADH); Enoyl-Acp
Reductase; Enoyl-Acp Reductase; Enoyl-Acyl Carrier
Protein; Enoyl-CoA Hydratase; Enoyl-CoA Hydratase, Mitochondrial;
Envelope Glycoprotein; Envelope Glycoprotein; Envelope glycoprotein
GP340; Envelope glycoprotein GP340/GP220; Eosinophil Cationic. . .
Regulator Protein; Fatty Acid/Phospholipid Synthesis Protei; Fatty
Acid-Binding Protein; Fatty Acid-Binding Protein, Adipocyte; Fatty
Acid-Binding Protein, Brain; Fatty acid-binding protein, liver;
Fatty aldehyde dehydrogenase; Fatty-Acid Amide Hydrolase; F-Box Only
Protein 2; Fc Fragment; Fc Gamma Receptor FCGR1-HUMAN; Feglymycin; Feline
Immunodeficiency Virus Protease; Feline Leukemia Virus Receptor-Binding
Domai; Ferredoxin; Ferredoxin II; Ferredoxin Reductase;
Ferredoxin: NADP+ Oxidoreductase; Ferredoxin: NADP+ Reductase;
Ferredoxin-Dependent Glutamate Synthase; Ferredoxin-NADp
Reductase; Ferredoxin-NADp Reductase; Ferredoxin-NADp+
Reductase; Ferredoxin-NADp+ Reductase; Ferric
Hydroxamate Receptor; Ferric Hydroxamate Uptake Receptor;
Ferrichrome-Binding Periplasmic Protein; Ferrichrome-Iron Receptor;
Ferrichrome-Iron Receptor Precursor; Ferripyochelin Binding Protein;
Ferripyoverdine Receptor;. . . protein 1A; Fk506-Binding Protein 4;
Fkbp12.6; FKBP12-rapamycin complex-associated protein; Fkbp25; Fkbp-Type
Peptidyl-Prolyl Cis-Trans Isom; Fksg76; FL cytokine receptor precursor;
Flavin reductase; Flavocytochrome B2; Flavocytochrome C;
Flavocytochrome C Fumarate Reductase; Flavocytochrome C3;
Flavocytochrome C3 Fumarate Reductase; Flavodoxin; Flavodoxin
Reductase; Flavohemoprotein; Flavoprotein; Fluorescent Protein
Fp538; Fmn-Binding Protein; Fms1 Protein; Focal Adhesion Kinase 1; Folate
receptor alpha; Folate receptor beta; Folate. . .
Fructose-Bisphosphate Aldolase A; Fructose-Bisphosphate Aldolase Class I;
Fructose-Bisphosphate Aldolase II; Ftsz; Fucose-Specific Lectin; Fumarase

C; Fumarate Hydratase Class II; Fumarate reductase flavoprotein subunit; Fumarylacetoacetate Hydrolase; Fusion Protein; Fusion Protein Consisting Of Kinesin-Like Pr; Fusion Protein Consisting Of Staphylococcus; Fv Fragment; G. . . .
 . . . ionotropic kainate 5; Glutamate Semialdehyde Aminotransferase; Glutamate-Cysteine Ligase; Glutamate-cysteine ligase catalytic subunit; Glutamate-cysteine ligase regulatory subunit; Glutaminase, kidney isoform; Glutaminase, liver isoform; Glutaminase-Asparaginase; Glutamine Aminotransferase; Glutamine Phosphoribosylpyrophosphate; Glutamine Phosphoribosylpyrophosphate Amidot; Glutamine Receptor 2; Glutamine Synthetase; Glutamyl-Endopeptidase; Glutamyl-tRNA Reductase; Glutamyl-tRNA Synthetase; Glutaredoxin 3; Glutaryl-Coa Dehydrogenase; Glutathine Synthetase; Glutathione Reductase; Glutathione reductase (mitochondrial); Glutathione S-Transferase; Glutathione S-Transferase; Glutathione S-Transferase 1-6; Glutathione S-Transferase 2; Glutathione S-Transferase 26 Kda; Glutathione S-Transferase A1; Glutathione S-Transferase. . . . Glutathione-S-Transferase; Glyceraldehyde 3-Phosphate Dehydrogenase; Glyceraldehyde 3-Phosphate Dehydrogenase; Glyceraldehyde 3-Phosphate Dehydrogenase A; Glyceraldehyde-3-Phosphate Dehydrogenase; Glyceraldehyde-3-Phosphate Dehydrogenase; Glyceraldehyde-3-phosphate dehydrogenase, liver ; Glyceraldehyde-3-phosphate dehydrogenase, testis-specific; Glycerol Dehydratase; Glycerol Dehydrogenase; Glycerol Kinase; Glycerol Uptake Facilitator Protein; Glycerol Uptake Operon Antiterminator-Re; Glycerol-3-Phosphate Cytidyltransferase; Glycerol-3-Phosphate. . . . Glycine receptor alpha-1 chain (Precursor); Glycine receptor alpha-3 chain; Glycine receptor beta chain; Glycogen phosphorylase; Glycogen Phosphorylase b; Glycogen Phosphorylase, Liver Form; Glycogen phosphorylase, muscle form; Glycogen Synthase 1; Glycogen Synthase Kinase-3 Beta; Glycogenin-1; Glycolate Oxidase; Glycolipid 2-Alpha-Mannosyltransferase; Glycolipid Transfer Protein;. . . . Alpha-Galac; Glycoprotein-Fucosylgalactoside Alpha-N-Ace; Glycosyl Transferase; Glycosylase; Glycosyltransferase A; Glycosyltransferase B; Glycosyltransferase Gtfa; Glycosyltransferase Gtfd; Glyoxalase Family Protein; Glyoxalase II; Glyoxylate reductase/ hydroxypyruvate reductase; Gmp Reductase I; GMP synthase (glutamine-hydrolyzing); Gmp Synthetase; Gomesin; Gonadotropin-releasing hormone II receptor; Gonadotropin-releasing hormone receptor; GP41 envelope protein (first heptad repeat);. . . . Histone-Lysine N-Methyltransferase, H3 Lysin; Hiv1 Gp41 Hser Analogue Peptide Ace-Ile-T; Hiv-1 Integrase; Hiv-1 Protease; HIV-1 Reverse Transcriptase; HIV-2 Protease; Hmg-CoA Reductase; Holliday Junction DNA Helicase Ruvb; Holliday-Junction Resolvase; Holo-; Holo-D-Glyceraldehyde-3-Phosphate Dehydrogen; Homo Sapiens V-Kit Hardy-Zuckerman 4 Feline;
 . . . Cluster Protein; Hydantoinase; Hydrogen peroxide-inducible genes activator; Hydrolase; Hydrolase Angiogenin; Hydroxyacid Oxidase 3; Hydroxyacylglutathione Hydrolase; Hydroxyethylthiazole Kinase; Hydroxylamine Oxidoreductase; Hydroxylamine Reductase; Hydroxymethylglutaryl-CoA lyase; Hydroxynitrile Lyase; Hydroxyquinol 1,2-Dioxygenase; Hydroxysteroid Sulfotransferase; Hypothetical 22.5 Kda Protein In Tub1-Cp; Hypothetical 22.5 Kda Protein In Tub1-Cpr3. . . . Bifunctional Enzyme; Iswi Protein; Kallikrein; Kallikrein 1; Kallikrein 6; Kanamycin nucleotidyltransferase; Kappa-4 Immunoglobulin; Kata Catalase; Kdo-8-Phosphate Synthetase; Kdpg Aldolase; Ketoacyl Reductase; Kex1; Killer Cell Immunoglobulin-Like Receptor 2Ds; Kindling Fluorescent Protein; Kinesin; Kinesin Heavy Chain; Kinesin Heavy Chain-Like Protein; Kinesin Motor Ncd;. . . . L; L-2-Haloacid Dehalogenase; L-2-Hydroxyisocaproate Dehydrogenase; L-3-Hydroxyacyl Coa Dehydrogenase; L-3-Hydroxyabyl-Coa Dehydrogenase; L-3-Phosphoserine Phosphatase; Laccase; Laccase; Laccase 1; Laccase 2; Lactadherin;

Lactaldehyde Reductase; Lactate Dehydrogenase; Lactate Dehydrogenase; Lactoferrin; Lactoferrin; Lactose Permease; Lactotransferrin; Lactoylglutathione Lyase; L-Alanine Dehydrogenase; L-Allo-Threonine Aldolase; Lambda Exonuclease; Laminarinase 16A; L-Amino. . . Leucyl-tRNA Synthetase; Leucyl-tRNA synthetase, cytoplasmic; Leukoagglutinin; Leukocidin F Subunit; Leukocyte Elastase; Leukosialin; Leukoptriene A-4 Hydrolase; Leukotriene B4 12-Hydroxydehydrogenase/Pros; Levansucrase; Levodione Reductase; L-Fucose Isomerase; L-Fucose 1-Phosphate Aldolase; L-Fucose-1-Phosphate Aldolase; L-Histidinol Dehydrogenase; light chain; . . . 2; Lipase 3; Lipase, Gastric; Lipid Transfer Protein; Lipj; Lipoate-Protein Ligase, Putative; Lipoprotein Mxim; Lipoprotein Nlpi; Lipoxygenase-3; Lipoyltransferase 1; Lithostathine; Liver Alcohol Dehydrogenase; Liver Carboxylesterase; Liver Carboxylesterase I; Liver Fatty Acid Binding Protein; Liver Glycogen Phosphorylase; L-Lactate Dehydrogenase; L-Lactate Dehydrogenase; L-lactate dehydrogenase A chain; L-lactate dehydrogenase A-like 6A; L-lactate dehydrogenase A-like 6B; L-lactate dehydrogenase. . . amino acid transporter 1 (LAT1); L-type amino acid transporter 2; Luciferase; Lumazine Synthase; Luteinizing Hormone Releasing Hormone (LHRH) Receptor; L-Xylulose Reductase; Lymphocyte function-associated antigen 1 (CD11a antigen); Lysine Biosynthesis Enzyme; Lysine hydroxylase; Lysozyme; Lysozyme C; Lysozyme Insertion Mutant With Ala Inserted; Lysozyme; Lysozyme. . . Product Hydrolase; Metallo Beta-Lactamase II; Metallochaperone Atx1; Methionine adenosyltransferase; Methionine Aminopeptidase; Methionine Aminopeptidase 2; Methionine Gamma-Lyase; Methionine Synthase; Methionine synthase reductase; Methionine-R-sulfoxide reductase; Methionine-R-sulfoxide reductase B2; Methionyl Aminopeptidase; Methionyl-tRNA synthetase; Methoxy Mycolic Acid Synthase 2; Methuselach Ectodomain; Methyl-Accepting Chemotaxis Protein; Methylaspartate Mutase S Chain; Methylated-DNA-protein-cysteine methyltransferase; Methylcrotonoyl-CoA; Methylcrotonoyl-CoA 2; Methylene Tetrahydromethanopterin Dehydrogen; Methylenetetrahydrofolate Dehydrogenase/Cy; Methylenetetrahydrofolate reductase; Methylglyoxal Synthase; Methylmalonate-semialdehyde dehydrogenase; Methylmalonic aciduria protein; Methylmalonyl Coa Decarboxylase; Methylmalonyl-Coa Carboxyltransferase 12S Su; Methylmalonyl-CoA mutase, mitochondrial precursor; Mevalonate Kinase;. . . Cd14; Mono-Heme C-Type Cytochrome Scya; Monomer Hemoglobin Component III; Monomer Hemoglobin Component IV; Monomeric Sarcosine Oxidase; Monomethylamine Methyltransferase Mtmb1; Morphinone Reductase; Motuporin; M-Phase Inducer Phosphatase 2; Mrell Nuclease; Mrna Capping Enzyme; Mrna Decapping Enzyme; Mrsd Protein; Mta/Sah Nucleosidase; Mu Class Glutathione. . . Dehydrogenase; Nad-Dependent Malic Enzyme; Nad-Dependent Malic Enzyme; Nad-Dependent Malic Enzyme, Mitochondria; NAD-dependent malic enzyme, mitochondrial precursor; Nadh Oxidase; Nadh Oxidase/Nitrite Reductase; Nadh Peroxidase; Nadh Pyrophosphatase; Nadh-Azoreductase, Fmn-Dependent; NADH-cytochrome b5 reductase; Nadh-Dependent Butanol Dehydrogenase; NADH-ubiquinone oxidoreductase 13 kDa-A subunit, mitochondrial precursor; NADH-ubiquinone oxidoreductase 13 kDa-B subunit; NADH-ubiquinone oxidoreductase 15 kDa subunit;. . . . NADP-dependent malic enzyme; NADP-dependent malic enzyme, mitochondrial precursor; Nadp-Dependent Mannitol Dehydrogenase; Nadp-Dependent Nonphosphorylating Glyceralde; Nadph Dehydrogenase 1; Nadph Dependent Thioredoxin Reductase; Nadph : Ferredoxin Oxidoreductase; Nadph-Cytochrome P450 Reductase; Nadph-Flavin Oxidoreductase; Nadp-Malate Dehydrogenase; Nagd Protein, Putative; Nalpj; Namn Adenylyltransferase; Nbla; N-Carbamyl-D-Amino Acid Amidohydrolase; Ndx1; Nei Endonuclease Viii-Like 1; Neocarzinostatin;. . . (ganglion) receptor; Nicotinic acetylcholine Receptor alpha2/alpha3; Nima-Related

Protein; Nine-Haem Cytochrome C; Nine-Heme Cytochrome C; Nit-Fragile Histidine Triad Fusion Protein; Nitrate Reductase; Nitric Oxide Reductase; Nitric Oxide Synthase; Nitric oxide synthase IIB; Nitric Oxide Synthase, Inducible; Nitric-Oxide Reductase Cytochrome P450 55A1; Nitric-Oxide Synthase; Nitric-oxide synthase brain; Nitric-Oxide Synthase Homolog; Nitric-oxide synthase IIC; Nitric-Oxide Synthase, Brain; Nitric-Oxide Synthase, Endothelial; Nitrite Reductase; Nitrogen Fixation Regulatory Protein Fixl; Nitrogen Regulation Protein; Nitrogen Regulatory lia Protein; Nitrogen Regulatory Protein Pii; Nitrogenase Iron Protein; Nitrogenase Iron Protein 1; Nitrophorin 1; Nitrophorin 2; Nitrophorin 4; Nitroreductase; Nitroreductase Family Protein; Nitrosocyanin; Nitrous Oxide Reductase; Nitrous-Oxide Reductase; Nk Receptor; NMDA receptor; N-Methyl-D-Aspartate Receptor Subunit 1; Nmra; Nogalonic Acid Methyl Ester Cyclase; Non Catalytic Protein 1; Nonaheme Cytochrome. . . . Penicillin-binding protein 4 precursor; Penicillin-Binding Protein 5; Penicillin-binding protein 5 precursor; Penicillin-binding proteins 1A/1B; Penicillin-Insensitive Murein Endopeptidase; Penicillopepsin; Pentaerythritol Tetranitrate Reductase; Penton Protein; Pentaerythritol Tetranitrate Reductase; Pept1; Peptaibol; Peptide; Peptide Amidase; Peptide Deformylase; Peptide Deformylase 2; Peptide Deformylase Defb; Peptide Deformylase Pdf1; Peptide Methionine Sulfoxide Reductase; Peptide N-Myristoyltransferase; Peptide Transporter Tap1; Peptide-N; Peptidic Toxin Nodularin; Peptidoglycan Recognition Protein I-Alpha; Peptidoglycan Recognition Protein Sa Cg11709; Peptidoglycan synthetase.

- . . . enzyme; Peroxisomal Carnitine O-Octanoyltransfer; Peroxisomal Carnitine O-Octanoyltransferase; Peroxisomal Hydratase-Dehydrogenase-Epim; Peroxisomal Hydratase-Dehydrogenase-Epimerase; Peroxisomal multifunctional enzyme type 2; Peroxisomal Trans 2-Enoyl Coa Reductase; Peroxisome Proliferator Activated Receptor A; Peroxisome Proliferator Activated Receptor D; Peroxisome Proliferator Activated Receptor G; pH 2.5 Acid Phosphatase; Phage. . . . G/H Synthase 1 Precursor; Prostaglandin G/H Synthase 2; Prostaglandin H2 Synthase; Prostaglandin H2 Synthase-1; Prostaglandin H2 Synthase-2; Prostaglandin Receptor; Prostaglandin-E2 9-Reductase; Prostate-specific membrane antigen (7E11-C5.3 antigen/FOLH1-human/glutamate carboxypeptidase 11); Prostatic Acid Phosphatase; Protease; Protease II; Protease Retropepsin; Protease Synthase and Sporulation Negati;. . . .
- . . . S; Proto-Oncogene Tyrosine-Protein Kinase Src; Protooncprotein; Protoporphyrinogen Oxidase, Mitochondria; P-Selectin; Pseudoazurin; Pseudocatalase; Pseudomonas Aeruginosa Lectin II; Psychrophilic Phosphatase I; Pteridine Reductase; Pteridine Reductase 1; Pteridine Reductase 2; Pts System, Chitobiose-Specific lib Comp; Pulmonary Surfactant-Associated Protein A; Pulmonary Surfactant-Associated Protein D; Pumilio 1; Pur Operon Repressor; Pure;. . . . Putative Flavin Oxidoreductase; Putative Glur0 Ligand Binding Core; Putative Glur0 Ligand Binding Core; Putative G-protein coupled receptor 40; Putative Ketoacyl Reductase; Putative Lipase From The G-D-S-L Family; Putative Mannosyl-3-Phosphoglycerate Phospha; Putative Modulator Of DNA Gyrase; Putative Nadph Dependent Oxidoreductases; Putative Oxalate. . . . Putative Protease La Homolog; Putative Riboflavin Kinase; Putative Snrnp Sm-Like Protein; Putative Sugar Kinase; Putative Transcriptional Regulator; Putative Xylanase; Putidaredoxin Reductase; Putrescine-Binding Protein; Pyelonephritic Adhesin; Pyranose Oxidase; Pyridoxal kinase; Pyridoxal Phosphate Biosynthetic Protein Pdx; Pyridoxal phosphate phosphatase; Pyridoxamine Kinase; Pyridoxine 5'-Phosphate. . . . Oxidase; Pyridoxine 5'-Phosphate Oxidase; Pyridoxine 5'-Phosphate Synthase; Pyridoxine-5'-phosphate oxidase; Pyrimidine Nucleoside Phosphorylase; Pyrogenic Exotoxin B; Pyrophosphatase; Pyrr Bifunctional Protein;

Pyrroline-5-carboxylate reductase 1; Pyrroline-5-carboxylate reductase 2; Pyrroline-5-carboxylate synthetase; Pyruvate carboxylase; Pyruvate Decarboxylase; Pyruvate dehydrogenase; Pyruvate Dehydrogenase E1 Component; Pyruvate dehydrogenase E1 component alpha subunit, somatic. . . . Pyruvoyl-Dependent Arginine Decarboxylase; Pyst1; Quercetin 2,3-Dioxygenase; Queuine tRNA-Ribosyltransferase; Quinohemoprotein Alcohol Dehydrogenase; Quinolinic Acid Phosphoribosyl Transferase; Quinolinic Acid Phosphoribosyltransferase; Quinone Oxidoreductase; Quinone Reductase; Quinone Reductase Type 2; Quinone-Reductase; Quinoprotein Ethanol Dehydrogenase; Rab GDP Disassociation Inhibitor Alpha; Rab6 Gtpase; RAC serine/threonine-protein kinase; Rac-Alpha Serine/Threonine Kinase; Radixin; RAF proto-oncogene serine/threonine-protein. . . . Mc; Ribonuclease Mcl; Ribonuclease Pancreatic; Ribonuclease pH; Ribonuclease Sa; Ribonuclease T1; Ribonuclease U2; Ribonuclease UK114; Ribonuclease Z; Ribonuclease, Seminal; Ribonucleoside-Diphosphate Reductase 2 Alpha; Ribonucleoside-Diphosphate Reductase M2 Chai; Ribonucleotide reductase; Ribonucleotide Reductase R2; Ribonucleotide Reductase R2-2 Small Subunit; Ribonucleotide Reductase Subunit R2F; Ribose 5-Phosphate Isomerase; Ribose-5-Phosphate Isomerase A; Ribose-5-Phosphate Isomerase Rpib; Ribosomal Protein L1; Ribosomal Protein L4; Ribosomal Protein S6. . . . Rop Ala2Ile2-6; Rubredoxin; Rubredoxin: Oxygen Oxidoreductase; Ruvb; Rv3303C-Lpda; Ryanodine receptor 1; S-Gamma86-Beta-Mercaptoethanol-Lysozyme; S-Gamma97-Beta-Mercaptoethanol Lysozyme; S100A6; S3-Rnase; Saccharopepsin; Saccharopepsin precursor; Saccharopine Reductase; S-Adenosylhomocysteine Hydrolase; S-Adenosyl-L-Homocysteine Hydrolase; S-Adenosyl-L-Methionine: Salicylic Acid Car; S-Adenosylmethionine Decarboxylase Proen; S-Adenosylmethionine Decarboxylase Proenzyme; S-Adenosylmethionine Synthetase; S-Adenosyl-Methyltransferase Mraw; Salicylic Acid-Binding. . . . Selenocysteine Lyase; Selenosubtilisin Bpn; Semaphorin 3A; Seminal Plasma Protein Pdc-109; Sensor Kinase Cita; Sensor Protein Fixl; Sensory Rhodopsin II; Sepiapterin Reductase; Serine Acetyltransferase; Serine Carboxypeptidase; Serine Hydroxymethyltransferase; Serine hydroxymethyltransferase (mitochondrial); Serine Hydroxymethyltransferase, Cytosolic; Serine palmitoyltransferase 1; Serine palmitoyltransferase 2; Serine Protease;. . . . 5-Dehydrogenase 2; Shikimate Kinase; Short Chain 3-Hydroxyacyl-CoA Dehydrogenase; Short chain 3-hydroxyacyl-CoA dehydrogenase, mitochondrial precursor; Short Chain Acyl-CoA Dehydrogenase; Short-Chain Dehydrogenase/Reductase Family M; Shp-2; Siah-1A Protein; Sialic Acid Binding Ig-Like Lectin 7; Sialidase; Sialidase 2; Sialoadhesin; Sigf1-Gfp Fusion Protein; Sigma Factor. . . .
 . . . A component B; Arylsulfatase A component C); sp P15531 NDKA-HUMAN Nucleoside diphosphate kinase A (EC 2.7.4.6); sp P16152 DHCA-HUMAN Carbonyl reductase (NADPH) 1; sp P16435 NCPR-HUMAN NADPH-cytochrome P450 reductase; sp P19099 C11B2-HUMAN Cytochrome P450 11B2; sp P19971 TYPH-HUMAN Thymidine phosphorylase; sp P20711 DDC-HUMAN Aromatic-L-amino-acid decarboxylase; sp P20813 CP2B6-HUMAN Cytochrome. . . . (EC 1.14.14.1); sp P21397 AOFA-HUMAN Amine oxidase (flavin-containing); sp P22309 UD11-HUMAN UDP-glucuronosyltransferase 1-1 precursor; sp P22310UD14-HUMAN UDP-glucuronosyltransferase; sp P23141 EST1-HUMAN Liver carboxylesterase 1 precursor (EC 3.1. 1.1); sp P27338 AOFB-HUMAN Amine oxidase B; sp P27707 DCK-HUMAN Deoxycytidine kinase (EC 2.7.1.74) (dCK);. . . . 1.1.1.1); sp P32320 CDD-HUMAN Cytidine deaminase (EC 3.5.4.5); sp P33261 CP2CJ-HUMAN Cytochrome P450 2C19 (EC 1.14.13.80); sp P42898 MTHR-HUMAN Methylenetetrahydrofolate reductase; sp P47989 XDH-HUMAN Xanthine dehydrogenase/oxidase; sp P48775 T230-HUMAN Tryptophan 2,3-dioxygenase (EC 1.13.11.11) (Tryptophan pyrrolase) (Tryptophanase) (Tryptophan oxygenase) (Tryptamin 2,3-dioxygenase) (TRPO);. . . .

(GDP-forming) beta-chain; Sucrose Phosphorylase; Sucrose-Specific Porin; Sugar Transport Protein; Sulfatase Modifying Factor 2; Sulfate Adenylyltransferase; Sulfide Dehydrogenase; Sulfite Oxidase; Sulfite Reductase; Sulfite Reductase Hemoprotein; Sulfolipid Biosynthesis; Sulfolipid Biosynthesis Protein Sqd1; sulfonyl urea receptor (SUR1); Sulfonylurea receptor 1; Sulfonylurea receptor 2; Sulfotransferase; Sulfotransferase Family, . . . Thiazide-sensitive sodium-chloride cotransporter; Thiazole Biosynthetic Enzyme; Thioesterase; Thioesterase I; Thiol Peroxidase; Thiol: Disulfide Interchange Protein; Thiol-Disulfide Oxidoreductase Resa; Thioredoxin; Thioredoxin reductase; Thiostrepton; Threonine Synthase; Threonine synthase-like 1; Threonyl-tRNA Synthetase; Threonyl-tRNA Synthetase 1; Thrombomodulin; Thrombospondin 1; Thromboxane A2 receptor; Thromboxane A2 synthase; . . .

. . . Trichodiene Synthase; Trichotoxin-A50E; Tricorn Protease; Trifunctional enzyme alpha subunit, mitochondrial precursor; Trigger Factor; Triggering Receptor Expressed On Myeloid Cel; Trihydroxynaphthalene Reductase; Trimethylamine Dehydrogenase; Triose Phosphate Isomerase; Triosephosphate Isomerase; Triosephosphate Isomerase, Glycosomal; Trk System Potassium Uptake Protein Trka Hom; tRNA; tRNA Cca-Adding Enzyme; tRNA Nucleotidyltransferase; tRNA-Guanine Transglycosylase; Tropinone Reductase-I; Tropinone Reductase-II; Troponin C, Slow Skeletal and Cardiac Muscles; Troponin I, cardiac muscle; Troponin T, cardiac muscle; Trp Operon Repressor; Trp Repressor Binding Protein Wrba; Trp RNA-Binding Attenuation Protein Complexed; Trypanothione Oxidoreductase; Trypanothione Reductase; Tryparedoxin II; Trypsin; Trypsin I; Trypsin II, Anionic; Trypsin Inhibitor Bgit; Trypsin Inhibitor I; Trypsin Iva; Trypsinogen; Tryptamine D receptors. . . Kinase Syk; Tyrosine-Protein Kinase Zap-70; Tyrosine-protein phosphatase, non-receptor type 1; Tyrosyl-tRNA Synthetase; Tyrosyl-tRNA synthetase, cytoplasmic; U1A RNA Binding Domain; Ubiquinol-cytochrome-c reductase complex core protein I, mitochondrial precursor; Ubiquitin; Ubiquitin-Activating Enzyme E1 1; Ubiquitin-Conjugating Enzyme E2 2; Ubiquitin-Conjugating Enzyme E2-25 Kda; Ubiquitin-Like. . . B12 Transport Protein Btuf; Vitamin D Binding Protein; Vitamin D Receptor; Vitamin D3 Receptor; Vitamin D-Dependent Calcium-Binding Prote; Vitamin K Reductase; Vitamin K-dependent protein Z; Voltage gated sodium channel; Voltage-dependent L-type calcium channel alpha-1C subunit; Voltage-dependent L-type calcium channel beta-1 subunit; . . . Xanthine-Guanine Phosphoribosyltransferase; Xanthosine Phosphorylase; Xenobiotic Acetyltransferase; Xylanase; Xylanase 10C; Xylanase Inhibitor Protein I; Xylanase Y; Xyloglucan Endotransglycosylase; Xylose Isomerase; Xylose Reductase; Xylose Reductase; Y177F Variant Of S. Enterica Rmla Bound To U; Yajq Protein; Ydr533C Protein; Yeast Cytochrome C Peroxidase; Yeast Iso-1-Ferrocycytochrome C; . . .

25. An apparatus comprising an X-ray source, and x-ray detector , a protein sample deposited on a substrate, the substrate having a thickness of between 20 nanometers and 25 microns, and. . .

. . . measuring a sample comprising: an X-ray source disposed so as to be able to expose the sample to X-rays; a detector disposed so as to be able to detect the X-ray fluorescence of the sample; a recording means for making a record of the X-ray fluorescence; and a record-control. . .

. . . measuring the X-ray fluorescence in the sulfur spectrum; measuring the amount of protein in the protein composition by measuring the absorbance of the light having a wavelength of about 260 nm; and calculating the relative ratio of the amount of sulfur. . .

L7 ANSWER 1 OF 4 IFIPAT COPYRIGHT 2010 IFI on STN

AB X-ray fluorescence (XRF) spectrometry has been used for detecting binding events and measuring binding selectivities between chemicals and receptors. XRF may also be used for estimating the therapeutic index of a chemical, for estimating the binding selectivity of a chemical versus chemical analogs, for measuring post-translational modifications of proteins, and for drug manufacturing.

=> d 3 ab

L7 ANSWER 3 OF 4 USPATFULL on STN

AB The present invention makes available methods and reagents for inhibiting cell growth or promoting cell differentiation comprising contacting the cell with a differegruline in a sufficient amount to inhibit cell proliferation or promote cell differentiation.

=> d 4 ab

L7 ANSWER 4 OF 4 USPAT2 on STN

AB The present invention makes available methods and reagents for inhibiting cell growth or promoting cell differentiation comprising contacting the cell with a differegruline in a sufficient amount to inhibit cell proliferation or promote cell differentiation.

=> d 4 kwic

L7 ANSWER 4 OF 4 USPAT2 on STN

SUMM . . . the model that the ZPA is responsible for normal anteroposterior patterning in the limb. The ZPA has been hypothesized to function by releasing a signal, termed a "morphogen", which forms a gradient across the early embryonic bud. According to this model, . . .

SUMM . . . one cell population is controlled by signals emitted from another. For instance, embryonic inductive signals are key regulatory proteins that function in vertebrate pattern formation, and are present in important signaling centers known to operate embryonically to define the organization of. . .

SUMM The natural function of the Ah receptor is unknown, however, deletion of the Ah receptor results in liver abnormalities and immune system impairment. Furthermore, the identification of any endogenous ligand has remained elusive, and how Ah receptor-mediated signaling. . .

DRWD . . . irradiation with 366 nm UV light. (D) Embryos irradiated with either long or short wavelength UV light following addition of frog-biliverdin IX α or (E) commercial biliverdin IX α .

DRWD FIG. 3. Biliverdin IX α rescues UV-irradiated embryos from dorsal axis deficiency. (A). Average DAI score of embryos irradiated with 254 nm UV light [gray] is 0.35. About 51% of embryos treated with biliverdin [black] are scored with a DAI between 5-4 with an average DAI score of 2.72. (B). Average DAI score of embryos irradiated with 366 nm UV light [gray] is 0.72. Nearly 55 % of embryos incubated with biliverdin [black] recover and are scored with 4-5 with an average DAI of 3.08 for the total population. The recoveries are. . . (C) The extent of embryo recovery from 254 nm [.quadrature.] or 366 nm [.circle-solid.] UV light irradiation is dependent on biliverdin concentration. Recovery was determined with the equation $R_{sub.i} = (x - uv) / (c - uv)$ where $R_{sub.i}$ = recovery index, x = average DAI for embryos incubated with biliverdin, uv = average DAI for

embryos exposed to UV and c=average DAI for control embryos.

DRWD FIG. 6. Physical chemical features of the HPLC fraction 23.3 min identifies it as biliverdin IX α . (A) The 23.3 min HPLC fraction has a unique UV-Vis absorption spectrum with characteristic peaks at 375 and 665. . . exchangeable protons ascertained by the mass increase in the presence of deuterium. These characteristics are identical to those of (1+) biliverdin. This identification was reinforced by the identical thin layer chromatography R.sub.. function. values (0.85 in a 3:1 chloroform: methanol mixture), co-chromatographic behavior of a commercial biliverdin sample and the yolk platelet material purified on C.sub.18 with HPLC and by superposition of both absorption and NMR spectra (see FIG. 7). [C] shows the structure of biliverdin and the numbering scheme used for NMR analysis. Note that the one-letter designators for the pyrrolic rings are different from. . .

DRWD FIG. 7. NMR one-dimensional .sup.1H spectrum and TOCSY spectra of the HPLC fraction 23.3 min identified as biliverdin IX α . (A) The one dimensional .sup.1H spectrum of the pure 23.3 min HPLC fraction is identical to that of commercial biliverdin IX α . Chemical shifts are relative to trimethylsilyl propionate at 0.00 ppm. .sup.1H NMR (Methanol-d.sub.4) δ 6.54(m, 1H,H-2.sup.1), 5.41(d, 1H,H-2.sup.2), 6.05(d,. . . the vinyl region from TOCSY spectrum of the oocyte molecule. The chemical shifts and coupling patterns are identical to commercial biliverdin IX α . Additionally, the coupling between the carbonyl carbon and the α and β methylene protons of the propionic acid side. . .

DRWD FIG. 8. Molecular switch for induction of dorsal axis. Biliverdin is proposed to interact with a cortical factor to trigger or switch-on the downstream activation of genes. When the chemical switch is turned ON, the Nieuwkoop center and the Spemann-Mangold organizer are sequentially formed. The UV irradiation of biliverdin renders an ineffective photo-product, the chemical switch remains OFF and the dorsalizing gene products that participate in the configuration of. . .

DRWD FIG. 9. Biliverdin arrests proliferation of HT29 colon cancer cells. Control HT29 cells incubated without biliverdin (.largecircle.). Within 1 day of exposure to biliverdin, proliferation is arrested (.circle-solid.). On day 30, treatment is discontinued. Twelve days later, proliferation resumes at a rate that is 43% of that of the control. The effects on proliferation by biliverdin purified from frog eggs are identical to those obtained with a commercial biliverdin preparation. Therefore, biliverdin is the active species in the egg preparation and not an undetected contaminant.

DRWD FIG. 10. Effect of biliverdin on proliferation of liposarcoma (LS), thyroid cancer (Th), B-lymphoblast (LB) and T-lymphoblast (LT) cells. Growth curves for treated cultures (.circle-solid.). . .

DRWD FIG. 12. Effect of biliverdin on alkaline phosphatase activity. Cellular alkaline phosphatase activity in treated cells (dark line) increases during the entire exposure to biliverdin compared to that of untreated control cells (thin line) that remain constant.

DRWD . . . analysis generates a high abundance signal of 583.2553 m/z. The chromatographic behavior and spectrometric results are identical to that of biliverdin standard. Biliverdin is present in all samples and accounts for their blue-green color.

DRWD FIG. 14. (a) Normalized values for oocyte volume and their biliverdin and zinc content at different stages of maturation. The ratios represent the value of the measured variable at any given. . . and volume values for each oocyte maturation stage were adapted from previous publications (Nomizu 1993, Tanabe 1974, Hausen 1991). The biliverdin content increases progressively during oogenesis

[.circle-solid.]. Its incremental accumulation correlates with that of zinc (.quadrature.) and volume [.gradient.]. (b) In the embryo, the biliverdin content [.circle-solid.] decreases steadily after fertilization. At stage 8, it is decreased to less than a half of the original. . . .

DRWD . . . a number of proteins that are resolved into distinctive peaks monitored at 280 nm (b) Only one fraction (arrow) contains biliverdin with its absorption peak at 375 nm and retains the blue-green color of the serum.

DRWD FIG. 18. Model of metabolic pathway of *Xenopus laevis* biliverdin . (a) Estrogen induces the hepatocytes to synthesize vitellogenin. Since biliverdin is a constituent of this protein, estrogen must induce the synthesis of the former as well. Once the tetrapyrrole is incorporated into vitellogenin, the complex is excreted into and transported in the frog plasma. Biliverdin is the signal molecule and vitellogenin is the carrier. The carrier, vitellogenin, binds to receptors expressed on the cell membrane surface of oocytes and it is internalized with the signal molecule, biliverdin, inside. Once in the oocyte, vitellogenin is processed to lipovitellin/phosvitin complexes. These complexes aggregate in a modular arrangement in yolk platelets. Lipovitellin binds the biliverdin molecule. (b) After fertilization, biliverdin interacts with a cortical factor(s) to establish sequentially the dorsalizing centers (Falchuk 2001). We propose that biliverdin is released from lipovitellin to exert its action. Biliverdin in conjunction to the cortical factor(s) switch-on downstream events that determine the sequential formation of the dorsalizing centers, the Nieuwkoop center [NC] and the Spemann-Mangold organizer [Org]. (c) Time line for biliverdin production, storage and activity. The first phase of biliverdin production and export lasts from few hours to few days. It comprises induction by estrogen of the maternal hepatocyte, synthesis of the biliverdin-vitellogenin complex, release of the complex to the circulation, import by the oocyte and processing. The second phase is the storage of biliverdin bound to lipovitellin in the oocyte. It could last up to three or more years, that is over 99.99% of the molecule's existence. Biliverdin integrity has to be protected and guaranteed during all this time. The third phase is the time when biliverdin exerts its activity. It happens in the first cell cycle of the embryo (usually in the first 100 minutes post fertilization). Biliverdin acts in a different organism than the one where the molecule originated.

DETD . . . system for isolation and identification of these master chemical signals while the embryo itself provides the means to test their function(s).

DETD We have now discovered that biliverdin is the dorsalizing cytoplasmic determinant in *X. laevis* oocytes. The present invention therefore relates to compositions of biliverdin, or derivatives thereof as defined by Formula I, which modulate cell growth, such as by modulating cell proliferation and cell. . . . present invention is also directed to methods for inhibiting cell proliferation or promoting cell differentiation to regulate the repair and/or functional performance of a wide range of cells, tissues and organs. For instance, the subject method has therapeutic and cosmetic applications. . . . regulation of neural tissues, bone and cartilage formation and repair, regulation of spermatogenesis, regulation of smooth muscle, regulation of lung, liver and other organs arising from the primitive gut, regulation of hematopoietic function, regulation of skin and hair growth, etc. Moreover, the subject methods can be performed on cells which are provided in. . . .

DETD The term "differeguline" refers to an agent which is capable of modulating cell proliferation or cell differentiation. Preferred differegulines are biliverdin, bilirubin and substituted

derivatives thereof.

DETD . . . the transcriptional activity of a target gene (i.e., a gene associated with the specific DNA sequence) is modulated as a function of the ligand bound to the receptor. Also, see Heyman et al., Cell, 68: 397-406 (1992), incorporated herein by reference.

DETD In certain embodiments, the compound is biliverdin. In certain other embodiments, the compound is bilirubin.

DETD . . . any animal. By any animal is meant any multicellular animal which contains nervous tissue. More particularly, is meant any fish, reptile, bird, amphibian or mammal and the like. The most preferable donors are mammals, especially mice and humans.

DETD . . . Brain areas of particular interest include any area from which progenitor cells can be obtained which will serve to restore function to a degenerated area of the host's brain. These regions include areas of the central nervous system (CNS) including the.

DETD . . . present invention makes use of differegulines for controlling the development of stem cells responsible for formation of the digestive tract, liver, lungs, and other organs which derive from the primitive gut. Therefore, for example, differegulines of the instant method can be employed for regulating the development and maintenance of an artificial liver which can have multiple metabolic functions of a normal liver. In an exemplary embodiment, the subject method can be used to regulate the proliferation and differentiation of digestive tube stem. . . .

DETD . . . embodiment, therapeutic compositions of differegulines can be utilized in conjunction with transplantation of such artificial livers, as well as embryonic liver structures, to regulate uptake of intraperitoneal implantation, vascularization, and in vivo differentiation and maintenance of the engrafted liver tissue.

DETD . . . to regulate such organs after physical, chemical or pathological insult. For instance, therapeutic compositions comprising differegulines can be utilized in liver repair subsequent to a partial hepatectomy.

DETD The methods and compositions of the present invention may be used as part of a regimen for restoring cartilage function to a connective tissue. Such methods are useful in, for example, the repair of defects or lesions in cartilage tissue. . . .

DETD . . . formed from polymers such as polyglycolic acid, polylactic acid, agarose gel, or other polymers which degrade over time as a function of hydrolysis of the polymer backbone into innocuous monomers. The matrices are designed to allow adequate nutrient and gas exchange. . . .

DETD . . . with a differeguline in order to actively remodel the implanted matrix and to make it more suitable for its intended function. As set out above with respect to tissue transplants, the artificial transplants suffer from the same deficiency of not being. . . .

DETD . . . be used as a contraceptive. In similar fashion, differegulines of the subject method are potentially useful for modulating normal ovarian function.

DETD Despite significant progress in reconstructive surgical techniques, scarring can be an important obstacle in regaining normal function and appearance of healed skin. This is particularly true when pathologic scarring such as keloids or hypertrophic scars of the. . . .

DETD Broadly, in one embodiment, this invention provides agonist and antagonist therapeutics, which can either mimic, potentiate or antagonize differegulin function, e.g., modulation of cellular differentiation and/or proliferation. The antagonist therapeutics of the invention are those therapeutics which antagonize, or inhibit, a differegulin function. Such antagonist therapeutics are most preferably identified by the assays described herein or by use of known

convenient in vitro assays, e.g., based on their ability to modulate and/or inhibit the interaction between a differegulin, such as biliverdin or a derivative thereof as defined by Formula I, and a receptor therefore, such as an aryl hydrocarbon receptor. In a preferred embodiment, the antagonist therapeutic is a biliverdin or a derivative thereof as defined by Formula I. It should be noted that in certain instances, an antagonist therapeutic. . .

DETD . . . differegulin or the interaction between a differegulin and a receptor therefore. Such agonist therapeutics include, but are not limited to, biliverdin or derivatives thereof as defined by Formula I.

DETD Identification of Biliverdin as the Dorsalizing Cytoplasmic Determinant

DETD . . . the dorsal axis. DCD is stable in organic solvents and destroyed by ultraviolet (UV) light. We have now discovered that biliverdin is the UV-sensitive molecule from *Xenopus laevis* oocytes that fulfills criteria for the long sought DCD. Stage 1 embryos exposed. . . to 0.4 time fraction of their first cycle inactivates the cytoplasmic determinant. At either wavelength, the embryos are depleted of biliverdin and are fated to develop dorsal axis deficiency. In contrast, UV-irradiated embryos subsequently incubated with oocyte or commercially available biliverdin in μM amounts recover to form dorsal axial structures. In contrast, incubation with either in vitro photo transformed biliverdin or biliverdin IX α dimethyl ester does not induce recovery.

DETD . . . or nucleic acid constituents of the egg or embryo. According to the present invention, the UV sensitive cytoplasmic factor is biliverdin. It is present in the oocyte, egg and embryo cytoplasm, is photo transformed by both short and long wave UV. . .

DETD . . . the UV-light exposure was applied well within the period of maximum effectiveness of UV light , in this case between T.sub.. function.m=0.3-0.4 (T.sub..function.m is the normalized time scale with a value of 1 representing the period from fertilization to the first mitosis). The. . .

DETD . . . 5 min, 0-100% B linear gradient from 5 to 45 min, 100% B from 45 to 60 min. The eluate absorbance was recorded at a range of wavelengths from 250 to 550 nm by means of a diode array.

DETD . . . of intact yolk platelets. The wavelength was selected on the basis of the absorption spectra of the target fraction. The absorbance change was monitored at 375 nm. The resultant photo transformation product was then re-chromatographed by HPLC. For comparison, a separate. . .

DETD The UV-sensitive fraction in the extracts studied is shown here to be biliverdin IX α , a substance that can be obtained commercially. Therefore, it was possible to analyze its biological activity with a fraction purified from oocytes or its commercially available counterpart and compare them to the effects of biliverdin photo transformed in vitro or of biliverdin dimethyl ester hydrochloride with its modified propionic side chains. Biliverdin IX α and derivatives were obtained from Porphyrin Products, Inc (Logan, Utah). Commercially available biliverdin IX α was subjected to the above extraction and chromatographic procedure beginning with the ethyl acetate step. The dimethyl ester required only HPLC purification. Photo transformed biliverdin was obtained by irradiating an aliquot of embryo culture solution containing biliverdin at the targeted concentration with 366 nm UV light for 12 h. The photo transformation of the biliverdin was verified spectrophotometrically by loss of the 375 nm absorption peak.

DETD The biological activities of biliverdin and its derivatives were tested by adding each of them to the incubation solution of embryos after the termination of the UV light exposure to either 254 or 366 nm

UV light and at selected time periods between T .sub..function
 .fm=0.4-2.5. Final concentrations of biliverdin ranged from
 0.05 to 5 μ M in less than 1% ethanol. The in vitro photo transformed
 biliverdin or the biliverdin dimethyl ester
 hydrochloride were added at a final concentration of 2.2 and 3.7 μ M,
 respectively. An extinction coefficient of 51,000. . .

DETD . . . methanol. The methanol solution was chromatographed on Sephadex
 LH-20 (0.9+18 cm) column. One ml fractions were collected and
 their UV absorbance monitored. The fractions with
 characteristic absorbance maximum at 379 nm were pooled, dried
 and suspended in 10% acetonitrile solution. The constituents were
 separated by HPLC using. . . 250+4.6 column (Phenomenex) and
 chromatography station (Waters) equipped with an automatic injector, in
 line vacuum pump, automatic gradient controller and absorbance
 detector. Buffer A was composed of 10% acetonitrile in ammonium acetate
 3 mM, pH 6.5. Buffer B was acetonitrile 100%.. . .

DETD . . . Products, Inc., San Gabriel, Calif.) as described and then
 incubated in the presence of pure candidate DCD or commercially
 available biliverdin (Sigma-Aldrich, St. Louis, Mo.) at final
 concentrations from 0.05 to 1.2 μ M in less than 1% ethanol. The
 concentrations were. . .

DETD A chromatography station (Waters) equipped with an automatic injector,
 in-line pump, automatic gradient controller and absorbance
 detector was used for reversed-phase HPLC. The extracts were dissolved
 in 1 ml of solvent A (20% acetonitrile, 3 mM. . .

DETD . . . prior to Fourier transformation. Proton NMR assignments were
 confirmed according to published methodology, and by comparison to
 spectra of commercial biliverdin IX α . All assignments
 refer to the numbering scheme in FIG. 6C.

DETD The one-dimensional .sup.1H spectrum is consistent with that of
 biliverdin IX α in terms of chemical shift distribution,
 and number of protons as determined by integration. The α , β ,
 γ and δ isomers of biliverdin are easily
 identified by differences in chemical shifts using the observations of
 Bonnett and McDonagh. For example, only the alpha. . . carboxyethyl
 side chains. This pattern is clearly evident in both the molecule of
 interest, and in the spectrum of commercial biliverdin
 IX α . The vinyl protons were identified from analysis of
 two-dimensional TOCSY, and DQFOSY spectra. ##EMI-00003##

DETD . . . 300A 250+4.6 column (Phenomenex). The first system used
 consisted of a Water Model 6000A solvent delivery system, Waters Model
 440 absorbance reader, and a Waters automated gradient
 controller. A number of gradients profiles were used in this system. In
 all cases,. . .

DETD . . . it and reduces its absorption comparable to the in vivo
 observation. The material in that fraction is identified unambiguously
 as biliverdin IX α by UV-Vis, mass and NMR spectrometry
 (FIGS. 6 and 7).

DETD To demonstrate the correlation between biliverdin photo
 transformation and dorsal axis deficiency, the UV exposed embryos were
 incubated with the intact tetrapyrrole. The DAI score of. . . with a
 DAI of 0. The degree of recovery of dorsal axis formation achieved by
 incubating embryos with commercially available biliverdin is
 comparable (FIG. 2E). This effect of biliverdin pertains to
 embryos irradiated with either 254 or 366 nm UV light (FIG. 3). It is
 concentration dependent since greater amount leads to greater degrees of
 recovery with a plateau of recovery is reached at 1.2 μ M
 biliverdin (FIG. 3C). In contrast, there is no recovery with 2.2
 μ M photo transformed biliverdin or 3.7 μ M
 biliverdin dimethyl ester hydrochloride (not shown). The time
 during development when biliverdin rescues irradiated embryos
 is maximal during the period encompassed by the first cleavage

(normalized time, fertilization-first mitosis $T_{m=1}$). The effectiveness decreases rapidly by $T_{m=1.75}$ and disappears by 3.

DETD Intact biliverdin, photo transformed or dimethyl ester are not dysmorphogens. When control fertilized oocytes unexposed to UV light are incubated with any. . . at least one of the carboxyl groups of its propionic side chains contribute to its biological activity since photo transformed biliverdin or dimethyl ester biliverdin do not induce irradiated embryos to form dorsal structures.

DETD The restoration of the capability of irradiated embryos to form a normal dorsal axis by addition of biliverdin together with the absence of multiple ectopic axes (FIGS. 2D, 2E) suggests that while both 254- and 366 nm UV light affects the biliverdin in the cytoplasmic yolk platelet, neither affect the localization of the cortical determinant to the future dorsal zone. A single normal dorsal axis in biliverdin-rescued embryos can only take place if the cortical determinant is properly localized to the dorso-vegetal zone. Currently, it is believed. . . identical adorsal teratology produced by either 254 or 366 nm UV irradiation together with the rescue of dorsal axes by biliverdin, suggest that the UV light perturbation of cortical rotation may be more complex, perhaps differ from the current model, and. . .

DETD Following sperm entry, the yolk platelets are concentrated to the entire vegetal hemisphere of the fertilized eggs. Biliverdin may be released from the organelles to interact with the cortical factor (FIG. 8). The biliverdin-cortical factor complex, localized to the future dorso-vegetal zone, can act as a switch-ON mode to initiate a cascade of events. . . transition (MBT) determine the configuration of the dorsal axis and inhibit the activity of other ventralizing signals. Photo transformation of biliverdin by UV light generates an ineffective product. Therefore, the chemical switch remains OFF, the Nieuwkoop center and the Spemann-Mangold organizer. . .

DETD Commercially available biliverdin matches the dorsalizing activity of the isolated material. Thus, commercial biliverdin rescues embryos exposed to UV light from the expected dorsal axis deficiency. The spectrum of rescue is comparable to that obtained with oocyte-derived biliverdin.

DETD The adult frog liver, lung, and muscle contain a number of retinoids and precursors but do not contain biliverdine. The only adult tissue where. . .

DETD The biological effectiveness of biliverdin in driving the differentiation process forward applies to the known differentiation pathology characteristic of neoplastic adult cells. Thus, biliverdine arrests. . .

DETD Biliverdin causes human colon adenocarcinoma to accumulate p21^{sup.}(Cip1) and p27^{sup.}(Kip1), increase the number of cells in G₁ and arrest their proliferation when incubating the cells with biliverdin. Subsequently, the contents of the differentiation markers, alkaline phosphatase, carcinoembryonic antigen and triacyl glycerol, are markedly increased. The dimethyl biliverdin ester is inactive indicating the propionic side chains are essential for the effects. The inhibitory effect on proliferation also applies. . . two mouse lymphomas. Concurrently, triacyl glycerol is upregulated in liposarcoma cells and 3T3 fibroblasts. The proliferative arrests are reversed when biliverdin is removed.

DETD . . . Assay Kit (Bio-Rad). The aliquot was added to 800 and 190 μ l of Milli-Q water and Coomassie blue dye, respectively. Absorbance was detected at 595 nm on a Varian Cary UV-Visible Spectrophotometer. The absorbance was compared against a standard curve created with bovine serum albumin from 2.5 to 25 μ g/ml. Two hundred μ l of. . .

DETD Biliverdin IX α exerts powerful effects on human colon

cancer HT 29 cells. Normally, the contents of p21 and p27, known inhibitors. . . is activated, Rb protein is phosphorylated and they resume cycling in log phase proliferation. In contrast, addition of 4×10^{-7} M biliverdin to the incubation medium of the low-density cultures, results in persistence and/or progressive increase in p21 and p27 content starting. . . 16 hs. This effect on p21 induction is dependent on one or both of the propionic acid side chains of biliverdin. When the dimethyl ester biliverdin analog, with its blocked propionic acid side chains, is used, p21 becomes nearly undetectable, identical to that of a control culture that has entered log phase. The effects of biliverdin on p21 and p27 content in HT 29 colon cancer cells is summarized in the tables below.

DETD

TABLE IV

The effects of biliverdin on p 21 content in HT 29 Colon Cancer Cells

Treatment	0	16	30	40
No Biliverdin	+++	++	+	ND
(+) Biliverdin	+++	+++	+++	++++

DETD

TABLE V

The effects of biliverdin on p 27 content in HT 29 Colon Cancer Cells

Treatment	0	16	30	40
No Biliverdin	+++	++	+	ND
(+) Biliverdin	+++	+++	+++	++++

DETD . . . is still slower than that of the control cells that double every 18 hrs. Ten-fold higher amounts than the optimal biliverdin concentration, for example $5 \mu\text{M}$, also arrests proliferation though there is an associated decrease cell numbers in the first three days. In contrast, cellular survival or proliferation is not affected by amounts lower than 10×10^{-7} M biliverdin (not shown).

DETD . . . either secreted into the medium or remain within the cells are increased. Within the first three days of exposure to biliverdin , the amount of CEA secreted into the medium by HT 29 cells increases from that of its constitutive production of. . . to over 80 ng/ml/ 10×10^6 cells by days 9-12 (FIG. 11). Thereafter, CEA content in the medium decreases progressively even though biliverdin is still present in the medium. Beyond day 45, when cell division resumes, the CEA marker content returns to the. . .

DETD . . . alkaline phosphate activity is nearly constant during the entire study period (FIG. 12). By the sixth day of incubation with biliverdin, the enzyme activity increases progressively reaching a fifteen-fold peak by day 20. Biliverdin also induces an over expression of triacyl glycerol (Table VII). The cytoplasm becomes filled with fat droplets that visibly changes. . .

DETD Other cancer cells. Biliverdin also affects the proliferative rate of liposarcoma, thyroid carcinoma and two lymphoblast cell lines (FIG. 10). The liposarcoma and lymphoblasts. . .

DETD

TABLE VI

Distribution of Cell Cycle Stages of HT 29 Cancer Cells, %

Conditions	G.sub.1	S	G.sub.2
Control	42	47	11
Biliverdin	63	30	7
Troglitazone. ^{sup} 26	67	13	16

Liposarcoma cells also accumulate triacyl glycerol in response to biliverdin. The extent of the accumulation is determined by the composition of the incubation media. Triacyl glycerol is induced in liposarcoma. . . alone. The amount increases when bovine pituitary extract is added with the insulin. The highest production is achieved, however, when biliverdin is combined with insulin plus bovine pituitary extract.

DETD
TABLE VII

EFFECT OF BILIVERDIN ON CELLULAR TRIACYL GLYCEROL
(TAG) CONTENT

Cell Type Change	TAG, mg/10 ⁶ cells	%
Colon Adenocarcinoma		
1) Control	68	--
2) Biliverdin (4 + 10 ^{sup.-7} M)	105	
154		
Liposarcoma		
1) Control	12.3	--
2) Insulin (5 µg/ml)	17.1	139
3) Insulin (5 µg/ml), Pituitary Extract (20 µg/ml)	20.7	
168		
4) Insulin (5 µg/ml), Pituitary Extract (20 µg/ml)	33.5	
272		
and Biliverdin (4 + 10 ^{sup.-7} M)		
Normal Fibroblast		
1) Control	9.3	--
2) Insulin (5 µg/ml)	28.3	304
3) Insulin (5 µg/ml) and Biliverdin (4 + 10 ^{sup.-7} M)	54.5	
586		

DETD . . . affect the timing for the fat accumulation. After 9 days of incubation with insulin and bovine pituitary extract, but without biliverdin, the fat droplets in the cells are small and scattered diffusely throughout the cell. These globules continue to enlarge to. . . most of the cytoplasmic space by day 14. In contrast, by day 7 the cells incubated in the presence of biliverdin already contain large, grouped and prominent droplets. While the triacyl glycerol content increases in both HT 29 and liposarcoma cells,. . . of the liposarcoma cells. 3T3-L1 fibroblasts differentiate into adipocytes in the presence of insulin (Table VII). In the absence of biliverdin, this hormone increases the triacylglycerol content of 3T3-L1 fibroblasts by 3-fold relative to control. When biliverdin is added, a progressively greater content of triacyl glycerol is achieved at day 9. The increase is dependent on the biliverdin concentration. At the highest concentration used, 4+10^{sup.-7} M, there is a 5.8-fold increase of triacyl glycerol content over that of. . . in the cells incubated with differentiation medium alone and is nearly as high as observed with troglitazone. At a lower biliverdin concentration, 10^{sup.-8} M, there is a 3.5-fold increase compared to control values. Remarkably, the amounts of biliverdin required to achieve the effects on proliferation, cell cycle and differentiation marker up-regulation, i.e. 10^{sup.-7}-10^{sup.-10} M, are the same as. . .

DETD Biliverdin is a biological active molecule capable of inducing differentiation on a broad number of targets including embryos and adult normal and malignant cells. This is a novel conclusion since biliverdin is considered to be a breakdown product of heme without a metabolic function. However, biliverdin is

present normally in the embryo, not as a byproduct of heme metabolism to be discarded once converted into bilirubin, but as a primary product synthesized in the maternal liver following estrogen stimulation, loaded onto vitellogenin, secreted into plasma, taken up by the oocyte and stored for years in the yolk platelets. Once fertilization has taken place, the biliverdin is used up within hours as a necessary pre-requisite to establishing a dorsal axis. This first indication that the tetrapyrrole has a function is now extended by the current findings and is supported by at least one other independent study. In that latter. . . exposed to TPA is the up-regulation of heme oxygenase 1 (HO-1), the enzyme that catabolizes the conversion of heme to biliverdin. As a consequence, the biliverdin content of TPA-exposed and differentiating cells is increased. The up-regulation of HO-1 appears to be a necessary step for induction of the differentiation since inhibition of the oxygenase activity by tin protoporphyrin, suppresses both the conversion of heme to biliverdin and the differentiation by TPA. These findings, together with the present results suggest, therefore, that the differentiation process produced by TPA needs to be examined in the context of the possible role of cellular biliverdin content as a mediating agent. The confirmation of this possibility has intriguing implications to the corresponding differentiating effect of hemin itself, a molecule that differs from heme, the precursor of biliverdin, only in the oxidation state of its iron. Since both hemin and biliverdin induce differentiation, we propose that it is the protoporphyrin molecular structure that is the active principle for both of them. Furthermore, the iron species in hemin, absent in biliverdin, is not necessary for hemin-induced differentiation.

DETD The molecular mechanism of action for these effects of biliverdin (and that of one of its possible precursor hemin) on cancer and normal cells is currently unknown. However, biliverdin may act as a ligand to one or more intracellular receptor(s) that then activate (or repress) many genes that are. . . and liposarcoma cells. These ligand dependent reactions encompass particular differentiation pathways yet to be fully elucidated. We already know that biliverdin does not use either the retinoid signaling system (RAR or RXR) or the peroxisome proliferator-activated receptor (PPAR γ) system. Therefore, if the biliverdin effect on cancer cells reported here is mediated by a receptor-activated mechanism, it is a hitherto unrecognized system that represents a novel differentiation pathway. The aryl hydrocarbon receptor is activated by biliverdin at the concentrations used here. Similarly, protoporphyrin IX and hemin appear to be endogenous ligands for mitochondrial benzodiazepine receptors. The search for the putative receptors that function in developmental and differentiation processes following binding to biliverdin is under active study.

DETD Other mechanisms both at the level of transcription and/or translation need to be considered. Biliverdin could act directly as an inhibitor of proteolytic or lipolytic processes that increase the amounts of varied cellular proteins and. . .

DETD Transport and Storage of Biliverdin

DETD Biliverdin is a constituent of vitellogenin and lipovitellin, and therefore, the material contained in the oocyte/egg/embryo originates in the maternal liver. Vitellogenin transports biliverdin in the maternal plasma and carries it into the oocyte. Biliverdin is stored for years as a complex within the yolk platelet protein lipovitellin. In contrast to this long period of storage during oogenesis, once the embryo is formed, biliverdin exerts its function within the first cell cycle. Then, the total content of biliverdin in the embryo decreases progressively in the

first five hours after fertilization and prior to the rnid blastula transition.

DETD The distribution of biliverdin within the egg was determined by establishing its presence in separated cell compartments. Freshly spawned eggs were dejellied and then. . . et al. 1995). Five egg fractions were separated into the following densities (in g/ml): <1.07, 1.08-1.15, 1.16-1.20, 1.21-1.26, 1.27-1.30. The biliverdin content of each fraction was analyzed after extraction with two volumes of the organic extraction solvent mix composed of 8. . . acetate, 1 part methyl acetate and 50 µg/ml butylated hydroxy toluene. The fraction that retained the green color characteristic of biliverdin was recovered and dried.

DETD . . . 510 HPLC pump and a Waters Automated Gradient Controller. The eluate was monitored at 340 nm with a Waters 440 Absorbance Detector and the data recorded with a Hewlett Packard 3390 Integrator using a binary solvent system. The initial solvent was ammonium. . . with a linear increment from 5 to 45 min, then 100% ending solvent from 45 to 60 min. A wavelength absorbance scan was performed on selected fractions with a Varian-Cary 50 Bio UV-Vis spectrophotometer. A control sample of previously purified commercial biliverdin (Sigma, St. Louis, Mo.) was treated similarly with organic solvents, chromatographed under the same conditions and used as a standard. . .

DETD The time course for biliverdin appearance and accumulation in oocytes during oogenesis and its utilization during early embryogenesis was examined. Oocytes at different stages of. . . EDTA 30 mM, ascorbic acid 30 mM, Tris 20 mM, pH 7.4. Oocyte and embryo homogenates were extracted and their biliverdin content analyzed as described above.

DETD . . . absorption at 375 nm was determined in selected fractions with a Varian-Cary 50 Bio UV-Vis spectrophotometer. The fractions with high absorbance at 375 nm were extracted with organic solvents using a ternary system consisting of one part of chloroform and two. . .

DETD The fractions with high absorbance at 375 nm were extracted with the same ethyl acetate/methyl acetate mixture as was carried out with the oocyte and. . . min from the lipovitellin and vitellogenin extracts were analyzed by mass spectrometry and compared with the spectrum of a commercial biliverdin standard sample that was treated previously in a similar way, by organic solvents and HPLC separation. FIG. 13.

DETD The presence of biliverdin in mature eggs allowed the examination of the time course of biliverdin accumulation in oocytes and utilization in embryos during oocyte maturation and embryogenesis, respectively. The tetrapyrrole is barely detectable in stage I-II oocytes but increases significantly and progressively in stages III-VI (FIG. 14), the so-called vitellogenic phases. These changes. . . volume and zinc content also increase during oogenesis and the curves of their incremental accumulation correlate closely with that of biliverdin (FIG. 14). This correlation suggests a possible common mechanism for their individual increases. We had previously demonstrated that zinc incorporation. . . increase both in size and density leading to an increase in oocyte volume (Danilchik 1987). Since the time course of biliverdin accumulation during oogenesis correlates to these other two variables it suggests that its accumulation in the oocyte also may be. . .

DETD This premise is now confirmed by the finding that biliverdin is an intrinsic component of vitellogenin. Subsequent to estrogen administration, vitellogenin synthesis is induced in the frog's liver and secreted into the blood stream. The normally yellow plasma acquires an intense green color. Protein components of the green. . . from purified vitellogenin. In both cases, the green chromophore had a retention time and spectral characteristics identical to those of

biliverdin extracted from oocytes and eggs (FIG. 13). Therefore, the presence of a biliverdin-vitellogenin complex in the serum of estrogen-stimulated frogs contributes to its green color.

DETD . . . fractionation of egg homogenates separates cytosol, mitochondria, light and dense yolk platelets, nuclei and peroxisomes (Montorzi, 1995). Analysis of the biliverdin content in these egg constituents demonstrates that the tetrapyrrole is found principally in layers with densities between 1.21 and 1.23 g/ml. These are the layers that concentrate and separate yolk platelets. Therefore, the majority of biliverdin is localized to yolk platelets. A smaller amount of biliverdin appears in the heavier fractions that typically contain peroxisomes and nuclei, but may also contain the heaviest and densest yolk. . . .

DETD In the yolk platelets, biliverdin is associated with lipovitellin. The yolk platelet proteins are solubilized with NaCl. Lipovitellin can be separated from phosvitin by treatment. . . . after ultracentrifugation. The pellet containing lipovitellin is green and exhibits absorption peaks at 375 nm and 665 nm characteristic of biliverdin. The phosvitin-containing ammonium sulfate supernatant is not green and does not absorb at this wavelength. Size exclusion chromatography on Sephacryl. . . .

DETD . . . vitellogenin, the oocytes and eggs. The UV-Vis .sub.200-1000 nm wavelength scan of this fraction demonstrated the characteristic absorption spectrum of biliverdin confirmed by its molecular weight of (+1) 583.2553. Both results also are identical to the characteristics of purified commercial biliverdin used as standard. Jointly, these results indicate that biliverdin is bound to lipovitellin in the yolk platelets.

DETD Whereas biliverdin increases progressively during oogenesis (FIG. 14), once the egg is fertilized, its content in the embryo decreases. From its maximum. . . .

DETD Biliverdin is linked intimately to that of vitellogenin, including its upregulation in the liver by estrogens, its secretion into the plasma, its uptake by oocytes and its processing in yolk platelets (FIG. 18). Vitellogenin. . . . vitellogenin are incorporated into the protein during its synthesis (Montorzi 1994, Montorzi 1995, Dolphin 1971, Wallace 1970). Vitellogenin also contains biliverdin IX α . As with the other intrinsic constituents, it is likely that the tetrapyrrole, is incorporated into the protein during its synthesis in the hepatocyte. This requires the generation of sufficient amounts of biliverdin to associate with the nascent vitellogenin. A point of departure to begin to understand how the tetrapyrrole might be formed and how its metabolism might be linked to that of vitellogenin is to review the available information on biliverdin biochemistry and place it in context with vitellogenin synthesis in the liver and processing in the oocyte (FIG. 18). In those species studied, biliverdin is formed as a product of heme breakdown in mononuclear phagocytes. In these cells, the microsomal enzyme heme-oxygenase catalyzes the. . . . oxidation of heme to α -OH-hemin with a ferric (Fe.sup.+3) cation (Tenhunen 1969, Ishizawa 1983). Then, in a subsequent non-enzymatic step, biliverdin is formed after the release of Fe.sup.+3 and a molecule of CO (King 1978). Biliverdin binds to albumin (Blauer 1975) and the protein-tetrapyrrole complex is internalized by hepatocytes expressing receptors for the protein (Ockner 1983). Once in the liver, biliverdin binds to ligandins and undergoes further processing (Wooley 1976). If these biochemical processes pertain to estrogen stimulated frogs, then the hormone might regulate heme breakdown directly. Alternatively, since in the frog, biliverdin is an essential metabolite, its formation cannot be considered to be solely a heme degradation product. Therefore, other pathways for making biliverdin in the liver may be

operative in the frog, including hitherto unrecognized synthetic ones.

DETD . . . the microsomal fraction (Sergeev 1975) and induce changes in the architecture subcellular organelles including the Golgi apparatus (Lewis 1976). Conceivably, biliverdin synthesis could be induced or favored by estrogen and vitellogenin could be modified post translationally to include the tetrapyrrole in its structure. In any case, once the biliverdin-vitellogenin complex is formed, the protein acts as the vehicle to transport biliverdin in the plasma from its site of origin, the liver, to its site of storage in the oocyte. Normally, the frog's plasma is yellow, but following high dose estrogen administration, it becomes green owing to the high amount of biliverdin-vitellogenin product induced by the over stimulation and secreted into the blood stream.

DETD Biliverdin is brought into the oocyte when the biliverdin-vitellogenin complex in the plasma is internalized by the oocyte after binding to membrane receptors on coated pits (Opresko 1987). Once. . .

DETD The biliverdin associated with vitellogenin is located in the domain that is processed into lipovitellin. This is consistent with finding that when yolk platelet proteins are solubilized and separated, the one that contains biliverdin is lipovitellin. The yolk platelets, therefore, become the storage site for biliverdin. The lipovitellin-tetrapyrrole complexes are stored in these organelles for several years, the period of time that it takes for an. . .

DETD A possible binding site of biliverdin to lipovitellin has been proposed. A series of studies conducted on lamprey lipovitellin by means of X-ray crystallography, .sup.31P .sup.2H. . . computer modeling, revealed a funnel shaped, lipid-rich cavity of 28,000 Å³ buried inside the LV complex. Tentatively, one molecule of biliverdin was modeled inside this cavity (Anderson 1998). Hydrophobic amino acid residues are somewhat uniformly distributed over the lipid cavity surface. . .

DETD A noteworthy implication for the presumed positioning of biliverdin in the bottom of the lipid cavity surrounded by a hydrophobic environment could be protection of its structural integrity from. . . hydrophilic molecules. A neutron diffraction study carried out on lipovitellin supports this speculation. In the study, a negative signal was detected when using D₂O as a solvent and ascribed to represent the interior of the lipid cavity (Timmins 1992). Biliverdin is sensitive to variations in the redox state. Bilirubin is an immediate product after reduction of biliverdin, in a reaction that is catalyzed by biliverdin reductase (Schmid 1975, Seifried 1976). This reaction could take place non-enzymatically over the long time (up to several years) that yolk platelets are in storage in the maturing oocyte and slowly transform biliverdin. Considering the low aqueous/lipid partition coefficient of several compounds with redox activity and the characteristics of lipovitellin, this protein could provide an optimal chemical protection environment for biliverdin over the oocyte maturation period. The intracompartamental pH of yolk platelets has been estimated to be 5.7 during maturation and 5 or less during embryogenesis (Fagotto 1994). Therefore, given a pK_a of the propionic acid groups of biliverdin in that pH range (Lightner 1996), it may be predicted that in this chemical environment the molecule could be protonated. . .

DETD This model is consistent with the observation that even mild organic solvents can extract the biliverdin from lipovitellin and suggests a non-covalent binding of biliverdin to lipovitellin. This explanation could account for the easy extractability of biliverdin from lipovitellin and yolk platelets in our experiments and others (Redshaw 1971). While vitellogenin and its processed product lipovitellin, both contain biliverdin, the

conditions required to extract the tetrapyrrole from each protein differ, indicating the presence of distinct chemical environments. The organic solvent extraction protocol used to extract biliverdin from lipovitellin, whether as found within oocytes, yolk platelets or as the purified protein, fails to extract the green pigment from the parent vitellogenin. An alternative ternary system (chloroform, methanol and the aqueous sample) was required to extract biliverdin from vitellogenin as a pure molecule or as found in serum. This difference in requirements for organic solvent extraction suggests that the particular protein structure of vitellogenin and lipovitellin impacts the exposure of the biliverdin-carrying site to the surrounding solvent.

DETD Once the egg is fertilized, the embryo utilizes its stored biliverdin as one of the components required to generate a dorsal axis (Falchuk 2001). Toward that end, following fertilization the majority of the yolk platelets settle into the vegetal hemisphere of the embryo (Hausen 1991). This places yolk rich biliverdin in the region that will rotate toward the dorsal equatorial segment. Biliverdin then could be released in the operative dorsal region from its storage location in the lipovitellin complex during the narrow. . . window of time before the first mitosis that occurs about 70 to 90 minutes after fertilization. Subsequent to its release, biliverdin triggers a series of events that result in the formation of the dorsal axis in the embryo (Falchuk 2001). The lipovitellin structure and architecture must have evolved to respond to specific signal(s) by allowing biliverdin to act at the proper time and place following fertilization (FIG. 18).

DETD The release of biliverdin from lipovitellin could be accomplished through a number of possible mechanisms. One could be direct unloading from the lipid cavity. . . a transformation of the protein structure induced perhaps by an allosteric mechanism, could modify the cavity with subsequent release of biliverdin. Another possibility could be the release of biliverdin from the lipid cavity following proteolysis initiated immediately after fertilization. To date, no proteolytic enzyme has been demonstrated to exist. . . .

DETD Once released from its complex with lipovitellin, we propose that biliverdin interacts with a cortical factor(s) as a required step of the signaling cascade that determines the dorsal axis (Falchuk 2001), a critical event in morphogenesis. This important role of biliverdin in dorsal axis formation requires that its storage protein and vesicle be conserved. This is consistent with the observation that. . . vitellogenin and in particular the lipovitellin domain, is consistent with the expectation that this protein has an intrinsically critical biological function highly dependent on its sequence, its three-dimensional structure and its physico-chemical properties. This is confirmed in part by the finding. . . .

DETD In summary, oocytes require several years to mature prior to ovulation (Gilbert 2000). During that time biliverdin is stored in yolk platelets and must be protected from structural chemical modifications. In fact, biliverdin spends greater than 99.99% of its existence time in storage inside lipovitellin (FIG. 6). Biliverdin is a molecule of maternal extra-oocyte origin, imported and stored in the oocyte to act much later in a different organism, the embryo, immediately after fertilization to initiate morphogenesis. The finding of biliverdin bound normally by vitellogenin and lipovitellin increases the number of proteins known to associate with this tetrapyrrole. As already mentioned above, albumin is another example. In addition, the aryl hydrocarbon receptor (AhR) protein binds and is activated by biliverdin in the μM range (Phelan 1998). All of these findings are consistent with the view that biliverdin is a functional molecule.

=> s assay?(p)biliverdin and (birds or avian or reptil?)
L8 9 ASSAY?(P) BILIVERDIN AND (BIRDS OR AVIAN OR REPTIL?)

=> d 18 -19

L8 ANSWER 1 OF 9 CABA COPYRIGHT 2010 CABI on STN
AN 2008:77665 CABA
DN 20083067634
TI Eggshell pigmentation indicates pesticide contamination
AU Jagannath, A.; Shore, R. F.; Walker, L. A.; Ferns, P. N.; Gosler, A. G.
CS Edward Grey Institute, Department of Zoology, University of Oxford, South
Parks Road, Oxford OX1 3PS, UK. andrew.gosler@zoo.ox.ac.uk
SO Journal of Applied Ecology, (2008) Vol. 45, No. 1, pp. 133-140. 31 ref.
Publisher: Blackwell Publishing. Oxford
ISSN: 0021-8901
URL: <http://www.blackwell-synergy.com/loi/jpe>
CY United Kingdom
DT Journal
LA English
ED Entered STN: 4 Apr 2008
Last Updated on STN: 4 Apr 2008

L8 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2004:204056 CAPLUS
DN 140:213541
TI Assays for the detection of biliverdin in
birds and reptiles
IN Gregory, Christopher; Ritchie, Branson W.
PA University of Georgia Research Foundation, Inc., USA
SO PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	---	-----	-----	-----
PI	WO 2004020980	A2	20040311	WO 2003-US27134	20030827
	WO 2004020980	A3	20040624		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003260131	A1	20040319	AU 2003-260131	20030827
	US 20060252110	A1	20061109	US 2005-525893	20050708
PRAI	US 2002-406175P	P	20020827		
	WO 2003-US27134	W	20030827		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 3 OF 9 IFIPAT COPYRIGHT 2010 IFI on STN
AN 11303045 IFIPAT;IFIUDB;IFICDB
TI Assays for the detection of biliverdin in
birds and reptiles
IN Gregory Christopher; Ritchie Branson W

PA Unassigned Or Assigned To Individual (68000)
 PPA Georgia, University of Research Foundation Inc (Probable)
 PI US 20060252110 A1 20061109
 AI US 2003-525893 20030827 (10)
 WO 2003-US27134 20030827
 20050708 PCT 371 date
 20050708 PCT 102(e) date
 PRAI US 2002-406175P 20020827 (Provisional)
 FI US 20060252110 20061109
 DT Utility; Patent Application - First Publication
 FS CHEMICAL
 APPLICATION
 ED Entered STN: 9 Nov 2006
 Last Updated on STN: 19 Dec 2006
 CLMN 10

L8 ANSWER 4 OF 9 USPATFULL on STN
 AN 2010:91506 USPATFULL
 TI HEME OXYGENASE INHIBITORS, SCREENING METHODS FOR HEME OXYGENASE
 INHIBITORS AND METHODS OF USE OF HEME OXYGENASE INHIBITORS FOR
 ANTIMICROBIAL THERAPY
 IN Wilks, Angela, Baltimore, MD, UNITED STATES
 MacKerrel, JR., Alexander, Baltimore, MD, UNITED STATES
 Furci, Lena, Grove City, OH, UNITED STATES
 Lopes, Pedro, Baltimore, MD, UNITED STATES
 PA UNIVERSITY OF MARYLAND, BALTIMORE, Baltimore, MD, UNITED STATES (U.S.
 corporation)
 PI US 20100081661 A1 20100401
 AI US 2007-374964 A1 20070724 (12)
 WO 2007-US74233 20070724
 20091102 PCT 371 date
 PRAI US 2006-832892P 20060724 (60)
 US 2007-945710P 20070622 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 3230
 INCL INCLM: 514/243.000
 INCLS: 514/332.000; 514/411.000; 514/415.000; 514/563.000; 514/632.000;
 514/649.000
 NCL NCLM: 514/243.000
 NCLS: 514/332.000; 514/411.000; 514/415.000; 514/563.000; 514/632.000;
 514/649.000
 IC IPCI A61K0031-53 [I,A]; A61K0031-444 [I,A]; A61K0031-4427 [I,C*];
 A61K0031-403 [I,A]; A61K0031-405 [I,A]; A61K0031-195 [I,A];
 A61K0031-185 [I,C*]; A61K0031-155 [I,A]; A61K0031-135 [I,A]
 IPCR A61K0031-53 [I,C]; A61K0031-53 [I,A]; A61K0031-135 [I,C];
 A61K0031-135 [I,A]; A61K0031-155 [I,C]; A61K0031-155 [I,A];
 A61K0031-185 [I,C]; A61K0031-195 [I,A]; A61K0031-403 [I,C];
 A61K0031-403 [I,A]; A61K0031-405 [I,A]; A61K0031-4427 [I,C];
 A61K0031-444 [I,A]

L8 ANSWER 5 OF 9 USPATFULL on STN
 AN 2008:130932 USPATFULL
 TI Pharmaceutical compositions and therapeutic applications for the use of
 a synthetic vitamin B12 derivative, glutathionylcobalamin
 IN Brasch, Nicola E., Kent, OH, UNITED STATES
 Birch, Catherine Stephanie, Cheshire, UNITED KINGDOM
 Williams, John Henry Howatson, Corwen, UNITED KINGDOM
 PI US 20080113900 A1 20080515
 AI US 2007-901983 A1 20070920 (11)
 PRAI US 2006-846435P 20060922 (60)
 DT Utility

FS APPLICATION
LN.CNT 1910
INCL INCLM: 514/006.000
NCL NCLM: 514/006.000
IC IPCI A61K0038-06 [I,A]; A61P0025-00 [I,A]; A61P0025-28 [I,A];
A61P0027-06 [I,A]; A61P0027-00 [I,C*]; A61P0009-00 [I,A]
IPCR A61K0038-06 [I,C]; A61K0038-06 [I,A]; A61P0009-00 [I,C];
A61P0009-00 [I,A]; A61P0025-00 [I,C]; A61P0025-00 [I,A];
A61P0025-28 [I,A]; A61P0027-00 [I,C]; A61P0027-06 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 6 OF 9 USPATFULL on STN
AN 2008:87533 USPATFULL
TI Pharmaceutical compositions and therapeutic applications for the use of
a novel vitamin B12 derivative, N-acetyl-L-cysteinylcobalamin
IN Brasch, Nicola E., Kent, OH, UNITED STATES
Birch, Catherine Stephanie, Cheshire, UNITED KINGDOM
Williams, John Henry Howatson, Corwen, UNITED KINGDOM
PI US 20080076733 A1 20080327
AI US 2007-903066 A1 20070920 (11)
PRAI US 2006-846435P 20060922 (60)
DT Utility
FS APPLICATION
LN.CNT 1929
INCL INCLM: 514/052.000
NCL NCLM: 514/052.000
IC IPCI A61K0031-7042 [I,A]; A61P0025-28 [I,A]; A61P0025-00 [I,C*];
A61P0027-06 [I,A]; A61P0027-00 [I,C*]; A61P0009-00 [I,A]
IPCR A61K0031-7042 [I,C]; A61K0031-7042 [I,A]; A61P0009-00 [I,C];
A61P0009-00 [I,A]; A61P0025-00 [I,C]; A61P0025-28 [I,A];
A61P0027-00 [I,C]; A61P0027-06 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 7 OF 9 USPATFULL on STN
AN 2006:294944 USPATFULL
TI Assays for the detection of biliverdin in
birds and reptiles
IN Gregory, Christopher, 1015 Cooper Farm Road, Nicholson, GA, UNITED
STATES 30565
Ritchie, Branson W., Athens, GA, UNITED STATES
PI US 20060252110 A1 20061109
AI US 2003-525893 A1 20030827 (10)
WO 2003-US27134 20030827
20050708 PCT 371 date
PRAI US 2002-406175P 20020827 (60)
DT Utility
FS APPLICATION
LN.CNT 1196
INCL INCLM: 435/025.000
NCL NCLM: 435/025.000
IC IPCI C12Q0001-26 [I,A]
IPCR C12Q0001-26 [I,C]; C12Q0001-26 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 8 OF 9 USPATFULL on STN
AN 2002:301655 USPATFULL
TI Compounds and methods for regulating cell differentiation
IN Falchuk, Kenneth H., Newton, MA, UNITED STATES
PA President & Fellows of Harvard College, Cambridge, MA, UNITED STATES
(U.S. corporation)
PI US 20020169201 A1 20021114
US 6902881 B2 20050607

AI US 2001-8356 A1 20011113 (10)
 RLI Continuation-in-part of Ser. No. US 2001-977866, filed on 15 Oct 2001,
 PENDING
 PRAI US 2000-240497P 20001013 (60)
 US 2000-247299P 20001110 (60)
 US 2001-262233P 20010117 (60)
 US 2001-264814P 20010129 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 4893
 INCL INCLM: 514/422.000
 INCLS: 548/518.000
 NCL NCLM: 435/001.100; 514/422.000
 NCLS: 435/325.000; 514/359.000; 514/422.000; 548/518.000
 IC [7]
 ICM A61K031-4025
 ICS C07D043-14
 IPCI A61K0031-4025 [ICM,7]; C07D0043-14 [ICS,7]
 IPCI-2 A01N0001-00 [ICM,7]; A01N0043-38 [ICS,7]; A01N0043-34 [ICS,7,C*];
 C12N0005-02 [ICS,7]; A61K0031-409 [ICS,7]
 IPCR A61K0031-409 [I,C*]; A61K0031-409 [I,A]
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 9 OF 9 USPAT2 on STN
 AN 2002:301655 USPAT2
 TI Compounds and methods for regulating cell differentiation
 IN Falchuk, Kenneth H., Newton, MA, UNITED STATES
 PA President and Fellows of Harvard College, Cambridge, MA, UNITED STATES
 (U.S. corporation)
 PI US 6902881 B2 20050607
 AI US 2001-8356 20011113 (10)
 RLI Continuation-in-part of Ser. No. US 2001-977866, filed on 15 Oct 2001,
 PENDING
 PRAI US 2001-264814P 20010129 (60)
 US 2001-262233P 20010117 (60)
 US 2000-247299P 20001110 (60)
 US 2000-240497P 20001013 (60)
 DT Utility
 FS GRANTED
 LN.CNT 4994
 INCL INCLM: 435/001.100
 INCLS: 435/325.000; 514/359.000; 514/422.000
 NCL NCLM: 435/001.100; 514/422.000
 NCLS: 435/325.000; 514/359.000; 514/422.000; 548/518.000
 IC [7]
 ICM A01N001-00
 ICS A01N043-38; C12N005-02; A61K031-409
 IPCI A61K0031-4025 [ICM,7]; C07D0043-14 [ICS,7]
 IPCI-2 A01N0001-00 [ICM,7]; A01N0043-38 [ICS,7]; A01N0043-34 [ICS,7,C*];
 C12N0005-02 [ICS,7]; A61K0031-409 [ICS,7]
 IPCR A61K0031-409 [I,C*]; A61K0031-409 [I,A]
 EXF 435/1.1; 435/325; 514/359; 514/422
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d 18 9 kwic

L8 ANSWER 9 OF 9 USPAT2 on STN
 DETD . . . any animal. By any animal is meant any multicellular animal
 which contains nervous tissue. More particularly, is meant any fish,
 reptile, bird, amphibian or mammal and the like. The most
 preferable donors are mammals, especially mice and humans.

DETD . . . invention are those therapeutics which antagonize, or inhibit, a differegulin function. Such antagonist therapeutics are most preferably identified by the assays described herein or by use of known convenient in vitro assays, e.g., based on their ability to modulate and/or inhibit the interaction between a differegulin, such as biliverdin or a derivative thereof as defined by Formula I, and a receptor therefore, such as an aryl hydrocarbon receptor. In a preferred embodiment, the antagonist therapeutic is a biliverdin or a derivative thereof as defined by Formula I. It should be noted that in certain instances, an antagonist therapeutic. . . depending on the developmental history of the tissue being exposed to the therapeutic; preferably, suitable in vitro or in vivo assays, as described herein, may be utilized to determine the effect of a specific therapeutic and whether its administration is indicated. . . .

=> s 18 and reductase

L9 5 L8 AND REDUCTASE

=> d 19 1-5

L9 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2010 ACS on STN

AN 2004:204056 CAPLUS

DN 140:213541

TI Assays for the detection of biliverdin in birds and reptiles

IN Gregory, Christopher; Ritchie, Branson W.

PA University of Georgia Research Foundation, Inc., USA

SO PCT Int. Appl., 44 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004020980	A2	20040311	WO 2003-US27134	20030827
	WO 2004020980	A3	20040624		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003260131	A1	20040319	AU 2003-260131	20030827
	US 20060252110	A1	20061109	US 2005-525893	20050708
PRAI	US 2002-406175P	P	20020827		
	WO 2003-US27134	W	20030827		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 5 IFIPAT COPYRIGHT 2010 IFI on STN

AN 11303045 IFIPAT;IFIUDB;IFICDB

TI Assays for the detection of biliverdin in birds and reptiles

IN Gregory Christopher; Ritchie Branson W

PA Unassigned Or Assigned To Individual (68000)

PPA Georgia, University of Research Foundation Inc (Probable)

PI US 20060252110 A1 20061109

AI US 2003-525893 20030827 (10)

WO 2003-US27134 20030827

20050708 PCT 371 date

20050708 PCT 102(e) date

PRAI US 2002-406175P 20020827 (Provisional)

FI US 20060252110 20061109

DT Utility; Patent Application - First Publication

FS CHEMICAL

APPLICATION

ED Entered STN: 9 Nov 2006

Last Updated on STN: 19 Dec 2006

CLMN 10

L9 ANSWER 3 OF 5 USPATFULL on STN

AN 2006:294944 USPATFULL

TI Assays for the detection of biliverdin in
birds and reptiles

IN Gregory, Christopher, 1015 Cooper Farm Road, Nicholson, GA, UNITED
STATES 30565

Ritchie, Branson W., Athens, GA, UNITED STATES

PI US 20060252110 A1 20061109

AI US 2003-525893 A1 20030827 (10)

WO 2003-US27134 20030827

20050708 PCT 371 date

PRAI US 2002-406175P 20020827 (60)

DT Utility

FS APPLICATION

LN.CNT 1196

INCL INCLM: 435/025.000

NCL NCLM: 435/025.000

IC IPCI C12Q0001-26 [I,A]

IPCR C12Q0001-26 [I,C]; C12Q0001-26 [I,A]

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 4 OF 5 USPATFULL on STN

AN 2002:301655 USPATFULL

TI Compounds and methods for regulating cell differentiation

IN Falchuk, Kenneth H., Newton, MA, UNITED STATES

PA President & Fellows of Harvard College, Cambridge, MA, UNITED STATES
(U.S. corporation)

PI US 20020169201 A1 20021114

US 6902881 B2 20050607

AI US 2001-8356 A1 20011113 (10)

RLI Continuation-in-part of Ser. No. US 2001-977866, filed on 15 Oct 2001,
PENDING

PRAI US 2000-240497P 20001013 (60)

US 2000-247299P 20001110 (60)

US 2001-262233P 20010117 (60)

US 2001-264814P 20010129 (60)

DT Utility

FS APPLICATION

LN.CNT 4893

INCL INCLM: 514/422.000

INCLS: 548/518.000

NCL NCLM: 435/001.100; 514/422.000

NCLS: 435/325.000; 514/359.000; 514/422.000; 548/518.000

IC [7]

ICM A61K031-4025

ICS C07D043-14

IPCI A61K0031-4025 [ICM,7]; C07D0043-14 [ICS,7]

IPCI-2 A01N0001-00 [ICM,7]; A01N0043-38 [ICS,7]; A01N0043-34 [ICS,7,C*];
C12N0005-02 [ICS,7]; A61K0031-409 [ICS,7]
IPCR A61K0031-409 [I,C*]; A61K0031-409 [I,A]
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 5 OF 5 USPAT2 on STN
AN 2002:301655 USPAT2
TI Compounds and methods for regulating cell differentiation
IN Falchuk, Kenneth H., Newton, MA, UNITED STATES
PA President and Fellows of Harvard College, Cambridge, MA, UNITED STATES
(U.S. corporation)
PI US 6902881 B2 20050607
AI US 2001-8356 20011113 (10)
RLI Continuation-in-part of Ser. No. US 2001-977866, filed on 15 Oct 2001,
PENDING
PRAI US 2001-264814P 20010129 (60)
US 2001-262233P 20010117 (60)
US 2000-247299P 20001110 (60)
US 2000-240497P 20001013 (60)
DT Utility
FS GRANTED
LN.CNT 4994
INCL INCLM: 435/001.100
INCLS: 435/325.000; 514/359.000; 514/422.000
NCL NCLM: 435/001.100; 514/422.000
NCLS: 435/325.000; 514/359.000; 514/422.000; 548/518.000
IC [7]
ICM A01N001-00
ICS A01N043-38; C12N005-02; A61K031-409
IPCI A61K0031-4025 [ICM,7]; C07D0043-14 [ICS,7]
IPCI-2 A01N0001-00 [ICM,7]; A01N0043-38 [ICS,7]; A01N0043-34 [ICS,7,C*];
C12N0005-02 [ICS,7]; A61K0031-409 [ICS,7]
IPCR A61K0031-409 [I,C*]; A61K0031-409 [I,A]
EXF 435/1.1; 435/325; 514/359; 514/422
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d L9 4-5 ab

L9 ANSWER 4 OF 5 USPATFULL on STN
AB The present invention makes available methods and reagents for
inhibiting cell growth or promoting cell differentiation comprising
contacting the cell with a differegruline in a sufficient amount to
inhibit cell proliferation or promote cell differentiation.

L9 ANSWER 5 OF 5 USPAT2 on STN
AB The present invention makes available methods and reagents for
inhibiting cell growth or promoting cell differentiation comprising
contacting the cell with a differegruline in a sufficient amount to
inhibit cell proliferation or promote cell differentiation.

=> d 19 4 kwic

L9 ANSWER 4 OF 5 USPATFULL on STN
DETD . . . any animal. By any animal is meant any multicellular animal
which contains nervous tissue. More particularly, is meant any fish,
reptile, bird, amphibian or mammal and the like. The most
preferable donors are mammals, especially mice and humans.
DETD . . . invention are those therapeutics which antagonize, or inhibit,
a differegrulin function. Such antagonist therapeutics are most
preferably identified by the assays described herein or by use

of known convenient in vitro assays, e.g., based on their ability to modulate and/or inhibit the interaction between a differegulin, such as biliverdin or a derivative thereof as defined by Formula I, and a receptor therefore, such as an aryl hydrocarbon receptor. In a preferred embodiment, the antagonist therapeutic is a biliverdin or a derivative thereof as defined by Formula I. It should be noted that in certain instances, an antagonist therapeutic. . . depending on the developmental history of the tissue being exposed to the therapeutic; preferably, suitable in vitro or in vivo assays, as described herein, may be utilized to determine the effect of a specific therapeutic and whether its administration is indicated. . . .

DETD . . . the redox state. Bilirubin is an immediate product after reduction of biliverdin, in a reaction that is catalyzed by biliverdin reductase (Schmid 1975, Seifried 1976). This reaction could take place non-enzymatically over the long time (up to several years) that yolk. . . .

=> d hist

(FILE 'HOME' ENTERED AT 19:53:45 ON 29 APR 2010)

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 19:54:11 ON 29 APR 2010
SEA BILIVERDIN AND (AVIAN OR BIRD OR REPTIL?) AND (LIVER OR HEP

1 FILE CABA
1 FILE CAPLUS
1 FILE EMBASE
2 FILE IFIPAT
1 FILE MEDLINE
79 FILE USPATFULL
13 FILE USPAT2

L1 QUE BILIVERDIN AND (AVIAN OR BIRD OR REPTIL?) AND (LIVER OR HEP

FILE 'CABA, CAPLUS, EMBASE, IFIPAT, MEDLINE, USPATFULL, USPAT2' ENTERED
AT 19:58:09 ON 29 APR 2010

L2 98 S L1
L3 96 DUP REM L2 (2 DUPLICATES REMOVED)
L4 53 S L3 AND (HEPATIC OR LIVER) (P)FUNCTI?
L5 24 S L4 AND ABSORBANCE
L6 19 S L5 AND REDUCTASE
L7 4 S L6 AND DETECT?(P)BILIVERDIN
L8 9 S ASSAY?(P)BILIVERDIN AND (BIRDS OR AVIAN OR REPTIL?)
L9 5 S L8 AND REDUCTASE

=> s L9 and detect?(p)biverdin(p)birds(p)reptiles

L10 0 L9 AND DETECT?(P) BIVERDIN(P) BIRDS(P) REPTILES

=> d hist

(FILE 'HOME' ENTERED AT 19:53:45 ON 29 APR 2010)

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 19:54:11 ON 29 APR 2010
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1   FILE CABA
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FILE 'CABA, CAPLUS, EMBASE, IFIPAT, MEDLINE, USPATFULL, USPAT2' ENTERED
AT 19:58:09 ON 29 APR 2010

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L2      98 S L1
L3      96 DUP REM L2 (2 DUPLICATES REMOVED)
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L9       5 S L8 AND REDUCTASE
L10     0 S L9 AND DETECT?(P)BIVERDIN(P)BIRDS(P)REPTILES

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=> logoff

ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF

LOGOFF? (Y)/N/HOLD:y

COST IN U.S. DOLLARS

	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	135.31	140.36

STN INTERNATIONAL LOGOFF AT 20:06:58 ON 29 APR 2010